DISEASES OF CHILDREN

DISEASES OF CHILDREN

DESIGNED FOR THE USE OF STUDENTS
AND PRACTITIONERS OF MEDICINE

BY

HERMAN B. SHEFFIELD, M.D.

Formerly Instructor in Diseases of Children, New York Postgraduate Medical School
and Hospital, and Medical Director, Beth David Hospital, Consulting
Physician to the Jewish Home for Convalescents and the
East Side Clinic for Children.

*WITH 238 ILLUSTRATIONS, MOSTLY ORIGINAL,
AND NINE COLOR PLATES*

ST. LOUIS
C. V. MOSBY COMPANY
1921

Printed in the U. S. A.

TO THE MEMORY OF HIS BELOVED SON

ROBERT LEONTE

THIS VOLUME
IS AFFECTIONATELY DEDICATED
BY THE AUTHOR

PREFACE

This volume is the consummation of the author's experience in the field of pediatrics for nearly thirty years. It embodies the latest knowledge of the theory and practice of the diseases of infancy and childhood and is designed to meet the needs especially of the general practitioner and medical student.

The book is conveniently divided into fourteen sections, the classification of the diseases varying somewhat from that of older textbooks, so as to correspond to the modern conception of the causation of the diseases in question.

Infant feeding is based upon the most recent studies of the digestibility of proteins, fats and carbohydrates and upon the author's practical experience. The fads and fetichisms of the erratic reformer and senile reactionary are eliminated. Breast feeding is recommended in preference to bottle feeding, yet the author believes that hosts of perfectly healthy babies can be reared on cow's milk, if good judgment is applied in the selection of suitable milk mixtures. A mixed diet is advocated for infants over nine months of age and well tried formulas and diet lists are appended for infants and older children. A special dietary is also provided for mentally deficient children.

The author hopes that the chapter on examination of the patient and semeiology of disease will greatly aid especially the beginner to surmount the difficulties of diagnosis in infants and older children. The normal anatomy and physiology are contrasted with the abnormal. Prominent symptoms which several diseases have in common are analyzed in relation to their pathogenesis and the different methods of physical diagnosis are amply elucidated and illustrated. The article on the clinical significance of the large abdomen may prove of special interest to the reader.

The time for "snap diagnoses" is past. Every obscure fever can no longer be dubbed malaria, and every cold bronchitis, for the very good reason that up-to-date laymen are sufficiently educated to demand a more exact and scientific diagnosis. To meet this requirement, a careful survey is presented of the most modern methods of laboratory diagnosis, such as the Schick test, the complement-fixation reaction of tuberculosis, the tuberculin tests, the Wassermann reaction of syphilis, the Widal reaction of typhoid fever and the Weil-Felix reaction of typhus fever. The therapeutic value and the indications for the use of serums and vaccines

are fully discussed and the prophylactic efficiency of diphtheria-toxin-antitoxin-immunization is dwelt upon at length.

A mere glance at the chapter on materia medica will assure the reader that the author is not a therapeutic nihilist, but on the contrary, a firm believer in the efficiency of *some* drugs. One of the principal reasons for the survival of many, often utterly useless, proprietaries is the fact that medical students receive but perfunctory instruction in pharmacology and prescription writing, and often do not even appreciate the inertness and incompatibilities of the drugs contained in the concoctions. The author hopes that the young beginner will profit by memorizing his article on select and palatable medication and by making use of some of the numerous prescriptions distributed throughout the book. Attention is also directed to the instruction given in hydrotherapy, (which includes hypodermoclysis, saline, intravenous, intraperitoneal and intrasinus injections) massage, electricity, climatology and organotherapy.

To obviate the great loss of life that still prevails among the newborn, their diseases, and more especially those of septic nature, are gone into minutely. It has often occurred to the author that a certain number of cases of dislocation of the hip in infants instead of being congenital in character are in reality acquired as a result of sepsis, and he hopes that the reader will henceforth scrutinize these cases with greater care and possibly confirm the author's observations. Under the head of ''Feeble Vitality'' are grouped the diverse diseased conditions in which feeble vitality forms the predominating feature. Herein are included also the premature babies which need the special care chiefly to overcome their feeble vitality.

A separate chapter is also reserved for the numerous congenital malformations which are frequently amenable to treatment if taken in hand early and treated skillfully. This refers especially to congenital pyloric stenosis which is fully discussed from a medical as well as surgical point of view. A great many illustrations accompany the text and should prove helpful early to detect the divers abnormalities.

In the description of acute gastroenteric affections the classification of Finkelstein is followed with but slight modifications. As the great majority of these cases are the result of milk infection, stress is put chiefly upon the care in handling the milk and the dietetic changes that are indicated in individual cases. The active treatment is presented briefly and clearly and is based upon the author's personal experience. Faulty metabolism is treated in another section of the book, in which are grouped rachitis, scorbutus, acidosis, exudative diathesis, glycosuria and the allied affections. Intussusception and appendicitis are discussed as strictly surgical diseases.

The section on respiratory diseases includes those of the nose, throat, and ears. The importance of diseased tonsils and adenoids as the immediate cause of widespread systemic infections is given due prominence. Enucleation of the tonsils is advocated in preference to tonsillotomy, which latter generally fails to eradicate the source of the trouble. On the other hand, it cannot be denied that tonsillectomy is a rather serious operation. All the necessary precautions against untoward results are amply emphasized when speaking of the dangers of the operation. The diagnosis of deafness is elaborated with greater detail than in other text-books on pediatrics; the different tests will prove useful to the family physician who is often consulted about the listlessness and inattentiveness of some of his little patients. In the description of the diverse inflammations of the lungs and pleura, the author has endeavored to embody the very latest advances in serology which offer the only hope for the ultimate discovery of a specific against pneumonia. Influenza pneumonia is described in connection with influenza. The newest views on the etiology of asthma are given due consideration, but the author is inclined to the belief that the symptoms arising from protein sensitiveness, anaphylactic manifestations, etc., are entirely distinct from those of genuine asthma, and transient in character, irrespective of the therapeutic measures employed.

With introduction of school inspection in a number of states the fact has been disclosed that heart disease in children is by far more common than was generally supposed. How much of it may be congenital in character is difficult to judge from the reports at hand. In the section of heart disease, both the congenital and acquired forms of heart disease are fully elucidated. Rest and digitalis are still urged as the only reliable therapeutic means at our command in heart disease, more especially in the noncompensating variety. Unfortunately few children can accommodate themselves to a regime of everlasting rest. The benefits derived from graduated exercises in heart disease of children which are fully delineated in this chapter are as yet an unknown quantity, yet worthy of trial. The author has failed to find any signal diagnostic help from the use of the sphygmomanometer, sphygmograph, cardiograph, and similar apparatus, hence has omitted their description.

Except for the recent advances in the study of blood coagulation practically no progress has been made in the knowledge of the diverse blood affection. No attempt therefore has been made to disrupt the generally accepted classification and methods of treatment. For the want of a better term hemorrhea is used instead of hemophilia—which latter is an utterly inappropriate designation for spontaneous hemorrhage. Transfusion is recommended as the most reliable remedy to control this kind

of bleeding. The author has refrained from going into a minute description of the pathology of blood diseases, since excepting Von Jaksch's anemia, which is peculiar to childhood, they are fully discussed in text-books on general medicine.

To a great extent the same holds true of kidney diseases, except pyelitis, which is of very frequent occurrence in children, and hence is receiving careful attention. A great deal is yet to be learned about the management of pyelitis. Like the urinary antiseptics the use of vaccines and kidney flushing have thus far failed in the majority of recalcitrant cases. The importance of early diagnosis and prompt treatment of cervicitis and vulvovaginitis is strongly emphasized and should receive due consideration on the part of the general practitioner who has ample opportunities to observe them.

The rather frequent association of acidosis with pyelitis is deserving of consideration from an etiologic, as well as therapeutic, point of view.

In recent years the profession has learned to appreciate the vital rôle the ductless glands are playing in the human economy, and the need for further scientific investigation. This subject therefore is treated broadly, laying particular stress upon the diseases of the thyroid, thymus and pituitary glands. The correlation of the disturbed functions of these glands to mental deficiencies is emphasized in another section of the book, when discussing the mental affections of infants and older children. A separate chapter is devoted to this greatly neglected subject.

The diseases of the lymphatics, the skin and bones are dwelt upon at length. Attention may here be directed to the article on malignant disease in children which is often overlooked in the early stages. Several vivid photographs and roentgenograms illuminate the text.

The author hopes that the chapter on nervous diseases will be found especially instructive. The brain and cord have ceased to be organs best let alone, and the progressive surgeon does not at all hesitate to operate on the brain as occasions arise. Emphasis is put upon the advisability of operating upon suitable cases of cerebral hemorrhage in the newborn, spastic cerebral paralysis, epilepsy, etc. Spasmophilia and hysteria are elaborated with great care. In order to facilitate their study in connection with diseases of the brain as a whole, meningitis, poliomyelitis and encephalitis are incorporated in this chapter, although from an etiologic point of view they belong in the section of contagious diseases.

Of the greatest importance, of course, to the general practitioner is a thorough acquaintance with the communicable diseases of childhood which are ever rampant and creating an overabundance of misery to mankind. This subject is treated exhaustively, and includes a large number

of tropical diseases which have recently invaded our shores. A special article is allotted to pertussis in the newborn infant. Epidemic influenza in all its phases is described in detail, more especially its pathogenesis and serum treatment. The same applies to poliomyelitis already spoken of which is clarified by a large number of original illustrations. The exanthemata are discussed from a modern point of view. In the article on tuberculosis are included the tuberculous affections of the brain, glands, skin and bones. Syphilis, in all its forms, is receiving explicit consideration. As already stated, the diphtheria-toxin-antitoxin-immunization and the diverse laboratory diagnostic tests are discussed in the chapter on the prevention and control of disease.

In closing the author wishes to extend his gratitude to the authors and publishers whose literature and illustrations have aided him in the preparation of the book; and he is particularly grateful to his publishers for their liberal suggestions and good will.

H. B. S.

New York City.

CONTENTS

CHAPTER V

CHAPTER VI

CHAPTER X

CHAPTER XI

CHAPTER XII

COLOR PLATES

ILLUSTRATIONS

DISEASES OF CHILDREN

CHAPTER I

PREVENTION AND CONTROL OF DISEASE

NUTRITION AND INFANT FEEDING. HYGIENE AND SANITATION. IMMUNI-
ZATION. THERAPY

The warfare between health and disease evolves with the earliest inception of life of the organism. The battle is fiercely rampant and everlasting, the victory at best but temporary. Supremacy of health over disease fluctuates with the amount of inherent strength of the individual, the natural and acquired power of resistance, and the assistance received through prophylaxis and therapeusis.

Nature aims to exterminate the weak, and right at birth tests the vitality of the infant in a manner most hazardous to its subsistence. Thus, accustomed to the ideal domicile of the maternal uterus—protected from traumatism and atmospheric vicissitudes, nurtured without effort and animated without the touches of pain or distress—the newborn is suddenly cast upon its own resources into a sphere of eternal strife, where every organism, every element, is struggling for supremacy, and where the strongest—not invariably the fittest—triumphs.

Inherent Strength

Inherent strength is essential to active life, to maintenance of perfect health. A powerful constitution will overcome an attack of disease that will fell the weak and the frail. A strong organization will surmount hardships and rapidly recuperate after protracted illness. Inherent strength is not procurable after birth. It is a consummation, an inheritance, of ancestral virility and vigor, premarital purity, conjugal devotion, matrimonial chastity, sobriety and ideal hygiene. It can be fostered by regulation of marriage, conservative mutual selection, prohibition of consanguineous marriages and those encumbered by grave disease, habits, alcoholism and drug addictions, or extreme poverty. Finally, it can be greatly improved by judicious management of pregnancy.

Power of Resistance and Susceptibility

Immunity, protection, or power of resistance against disease, and to a slighter extent also susceptibility toward disease, may be natural or acquired. It varies in different individuals and in the same individual at different periods of life. Natural or congenital immunity is aptly exemplified by the comparatively rare occurrence of communicable diseases in infants under three months of age. Congenital susceptibility is demonstrable by the prevalence of certain affections in some families or races, *e. g.*, hemophilia, tuberculosis, amaurotic family idiocy and the like. In contrast to inherent vitality, acquired power of resistance is vastly influenced during the life of the child. Thus, immunity against communicable diseases is often temporarily or permanently conferred, naturally by a previous attack of the same malady (*e. g.*, yellow fever, pertussis), and artificially by: I. Suitable nutrition. II. Hygiene and sanitation. III. Immunization. IV. Drugs and physical therapeutic measures.

I. NUTRITION

Suitable nutrition is indispensable to the life and growth of the individual and to the maintenance and advancement of his power of resistance. The human economy demands for its sustenance a liberal supply of proteids (to build up and to reconstruct the tissues), fat and carbohydrates (to produce energy and heat), mineral salts (to help formation of bones and teeth), and water (to aid the solubility of the food elements and the excretion of waste products). An ideal food, therefore, must contain these five ingredients in more or less definite proportion, must be readily digestible and assimilable, and be free from pathogenic bacteria. (See Vitamines, p. 114.)

The Digestibility of the Proteins of Milk and Their Rôle in Infant Nutrition*

While in many respects our ideas of infant nutrition and feeding have been modified by the clinical and laboratory researches of the last ten or fifteen years, probably in no direction has the change of view been so marked as that regarding the proteins. It is not many years since the difficult digestion of cow's milk protein was looked upon as the important, probably the chief cause of our troubles in infant feeding, and many were the expedients resorted to, to overcome this

---

*In order to avoid repetition, and also to give the student the latest information on the subject, we are here abstracting the articles on "The Digestibility of the Proteins" by Dr. L. E. Holt, and "The Digestion of the Carbohydrates" by Drs. J. L. Morse and F. B. Talbot.

difficulty, such as various forms of diluent, peptonizing, the addition of sodium citrate, etc. But we have learned that the symptoms formerly ascribed to the proteins depend upon other conditions. The curds in stools we know are composed chiefly of fat; most of the colic and flatulence are due to carbohydrates, and constipation depends much more on fat and salts than on casein. All researches upon gastric digestion in infants agree that in practically all conditions pepsin is abundantly secreted. The use of such ferments in disturbances of digestion, though still widely resorted to, has no rational basis.

Modern practice has certainly been in the direction of using much higher proportions of protein than were formerly thought wise or safe. It is interesting, therefore, to inquire whether this custom is justified by our present knowledge of the digestion of protein by the infant; also whether it is advantageous or whether its use is fraught with some disadvantages or possible dangers not apparent on the surface.

Metabolism experiments made at the New York Babies' Hospital and in many other places have revealed the fact that under almost all circumstances infants possess a remarkable capacity for retaining nitrogen. Even in conditions of severe malnutrition, the protein of cow's milk is well borne, as shown by a positive nitrogen balance, even though the infants were losing weight. This capacity on the part of the infant to assimilate protein is an indication of how well nature has provided the means of replacing protein waste and promoting growth above other needs of the organism.

Clinical evidence of the infant's tolerance of protein is also not wanting. When the question is asked, What are the symptoms of protein indigestion or intolerance? we are compelled to reply that at present we cannot mention any definite symptom or group of symptoms which we can positively attribute to the proteins, in the sense that we can attribute other definite symptoms to the fats and to the carbohydrates.

Under these circumstances it is pertinent to inquire whether the present practice of giving much higher proteins than formerly is one to be recommended without reservation; is this safe? is it advantageous? or is it possibly injurious?

The protein needs of the body must certainly be provided for; but is it desirable to go much beyond this? Many elaborate calculations have been made to determine the actual protein needs of the infant. We think we are safe in assuming that they are supplied in woman's milk in sufficient amount, but in no considerable excess. The most recent analyses of Courtney and Fales show that during the mature period of lactation, i. e., after the first month, the average protein con-

tent of woman's milk is slightly less than 1.25 per cent. An infant taking cow's milk, as it is now usually modified, gets very much more than this.

To put it in another way, assuming an average composition of mature woman's milk to be 3.5 per cent fat, 7.5 per cent sugar, and 1.25 per cent protein, a nursing infant is receiving a little over 7 per cent of his calories in the form of protein; an average artificially-fed infant of three months, who is taking cow's milk one-half strength with sufficient sugar added to bring the carbohydrates up to 6 per cent, is receiving over 14 per cent of his calories as protein; while an average infant of six months, who is getting two-thirds milk with the same propor-tion of sugar, is receiving nearly 17 per cent of his calories in the form of protein. This is on the assumption that all the nitrogen in woman's milk and in cow's milk is alike available for nutrition, which is not quite true. While this is very nearly the case with cow's milk, woman's milk is known to contain nitrogen in other forms than protein (extractives, urea, etc.), which reduce the available nitrogen by nearly one-fifth. So that the discrepancy between the protein content of the two milks is even greater than at first appears.

Rubner has calculated that on the average the food of the infant should have 7 per cent of his calories in the form of protein. This we have already seen is practically that which is present in woman's milk. What then becomes of the excess of protein given in our common feed-ing mixtures? Our own metabolism experiments have shown that with high protein feeding there is at first a marked increase in nitrogen re-tention, but that this persists only for a short time and that increased intake is followed by an increased excretion which is nearly but not quite proportional.

Protein is needed first of all to supply the nitrogenous waste of the cells of the body, one of the constant phenomena of life; secondly for growth; and lastly it may supply heat. The waste or "wear-and-tear-needs" of the infant, as compared with the needs of the adult, are not great. Growth, according to Rubner, is not in proportion to the protein intake and cannot be increased above natural limits by increasing the protein intake. The excess he believes is simply burned in the body in the place of carbohydrate and fat. The inference from his observations is that the protein requirements of the infants are relatively small and that if taken in excess of this minimal requirement the surplus can be used up in the place of other food elements.

Some experimental evidence has been brought forward which in-dicates that we may not continue to increase the protein in the food without incurring some risks; that the protein of cow's milk when given

in considerable excess of the needs of the body may bring about disturbances of metabolism causing clinical symptoms of importance, even of gravity.

In a series of metabolism observations made four years ago at the Babies' Hospital, it was shown that if large amounts of protein of cow's milk were given without whey (i. e., without carbohydrates) certain definite symptoms regularly followed—prostration, fever and a leukocytosis, which symptoms ceased immediately upon resuming the ordinary diet.

Observations in Lusk's laboratory by Howland and also by Murlin and Hoobler have shown that an increase in the amount of protein given caused an immediate and very marked increase in the general metabolism, and also that if fat and carbohydrates were not furnished in the food in sufficient amount, the increased metabolism caused an actual loss of these substances from the tissues of the body. In Hoobler's case there was observed diarrhea and a condition of semistupor.

Symptoms like those mentioned in the foregoing observations have been seen, it is true, only when protein is given very much in excess of the amounts commonly employed. The cause of these symptoms is not yet understood and while the observations are by no means conclusive, they are strongly suggestive of possible harm which may result from very high protein feeding. The increase in the general metabolism from such feeding, and, under certain circumstances, the actual withdrawal of fat and carbohydrate from the body, may furnish an explanation of why it is so difficult to increase weight if fat and carbohydrate, but especially the latter, are much reduced.

Thus far we have considered the protein needs of the infant only from a quantitative standpoint, and until quite recent times this has been the chief subject of discussion. The only differences between proteins have been indicated by that somewhat vague term of "digestibility." The latest studies of the food proteins indicate that the amount of protein given is much less important than the nature of the protein furnished. We have learned from Abderhalden that our common food proteins are very complex substances, being made up of some sixteen or eighteen different amino-acids.

Osborne and Mendel have for years been carrying on an extensive series of feeding experiments upon animals to determine the specific value of the different amino-acids in nutrition. They have shown that certain amino-acids are indispensable for growth; others are relatively unimportant. Thus if gliadin, a wheat protein, be the form of protein given to the animal, although the animal may maintain its weight, no growth occurs. But if to this, without increasing the total protein,

a small amount of one of the amino-acids known as lysin is added, a gain in weight begins immediately and continues as long as lysin is administered; but it ceases at once when it is withheld, and begins when it is again furnished to the animal. From many such experiments they have reached the conclusion that lysin is indispensable for growth; without it, no matter what amount of protein food is given, the most that the animal can do is to maintain itself in equilibrium. There are three other amino-acids of great importance—cystin, tryptophan and glycocoll. It does not seem possible for normal nutrition to go on unless lysin, tryptophan and cystin are furnished in the food; glycocoll alone, there is good reason for believing, can be produced in the body by synthesis.

Now as to the bearing of this on infant feeding. Animal proteins, as a rule, are relatively rich in those amino-acids which we will call the essential ones, while many vegetable proteins are very deficient in them. But again there is a wide difference in the amino-acid content of the different animal proteins. Lactalbumin is the protein which contains the essential ones in largest proportion. Casein, however, is notably deficient in at least one important one, cystin. Mendel says if the supply of casein is limited, the curve of growth is altered, not for lack of total protein, which may be entirely adequate, but for lack only of cystin, for as soon as this is added to the food, normal growth at once begins. Growth, therefore, is limited by the supply of cystin. The deficiencies of casein are ordinarily made good by the amino-acids of lactalbumin. And right here it should be remembered, that in woman's milk the amount of lactalbumin is twice the casein, while in cow's milk it is only one-sixth the casein. It is surely not an accident that woman's milk has relatively twelve times as much lactalbumin as has cow's milk.

Woman's milk supplies not only the amount of protein needed by the infant for the first eight or nine months of life, it furnishes what is more important, the essential amino-acids in sufficient quantity. There are good reasons for believing that nature has not intended cow's milk to nourish even the calf for a very long period. The maximum secretion of cow's milk is reached at the beginning of the second month, after which it steadily declines. Moreover, the calf at birth usually has eighteen teeth, indicating again his early capacity for digesting other food than milk and also his need of it. Now, the secretion of woman's milk increases in quantity under normal conditions up to eight or nine months, and the human infant does not get his teeth until seven or eight months—a strong suggestion that up to this time other food is unnecessary for normal nutrition. But,

in the case of infants who are artificially fed, conditions are different; although the defects of cow's milk have not been wholly understood, we have lately seen the great advantage of the earlier use of other articles of diet—fruit juices, beef juice, egg, broth and even fresh vegetables. Thus we have been unconsciously doing what the calf has been doing for a very long time—supplying the deficiencies of cow's milk.

Returning to the subject of casein, Osborne and Mendel have found that the gain in weight with 9 per cent of the food solids in the form of casein was very low unless cystin was added, but if the casein was doubled or increased to 18 per cent, a normal rise in weight was seen. We have here, we believe, a important fact which sheds light on some of our failures and successes in infant feeding. We once thought that we were supplying the infant's protein needs when we gave as much protein in cow's milk as the protein in woman's milk. Evidently we were wrong. It now seems clear that some of our failures were not due to the fact that we were giving too much fat, but that we were not supplying in the protein given the amino-acids required for normal growth.

The success which has attended the use of formulas made from whole milk has not been entirely due to the fact that the fat disturbances have been avoided, but that in these formulas by greatly increasing the protein we have come much nearer supplying the infant's actual amino-acid needs of growth, especially in lysin and cystin. The excess of other protein food apparently is not injurious.

We have seen thus far (1) that the digestion of the protein of cow's milk is a much easier matter than was formerly supposed; (2) that while injury may without question be done by high protein feeding, this is very unlikely to occur, unless amounts much in excess of those commonly used in infant feeding are administered; (3) that in such amounts we have as yet neither clinical nor laboratory evidence to show that protein is harmful; (4) that although an infant receiving breast milk takes rather less than 7 per cent of his calories as protein, this cannot be taken as an exact criterion of how much protein should be administered when cow's milk is the food; (5) that the deficiency of cow's milk casein in certain essential amino-acids may be made up by giving an excess of this protein.

There remains for brief consideration the clinical use which may be made of these facts, not now in the feeding of healthy infants, but in the diet of those who are suffering from the most common forms of digestive disturbances—intolerance of fat or carbohydrates or both. The great advantages of high protein feeding and the extent to which

proteins are borne we have only recently appreciated. That an infant of four or five months could easily tolerate a milk mixture containing as much as 3.5 or even 4 per cent of protein has been to most of us a surprise, especially when the protein given is nearly all casein. In our experience, in acute intestinal disturbances it is the carbohydrates that are most frequently at fault, and sugars are even more badly borne than starches. Milk sugar seems then to cause more disturbance than any other form of carbohydrate. It is for such cases that Finkelstein's milk modification—best translated into English as protein milk (q. v.)—is so valuable. Its usefulness is seldom enhanced by preparing it from skimmed milk, but, in my experience, rather the contrary. For its relatively high fat is usually tolerated without difficulty when low sugar is given. This preparation is to be regarded as a therapeutic agent, not a method of infant feeding, but it is one of the most valuable additions to our resources that has been made in recent years.

Physiology and Pathology of the Digestion of the Carbohydrates in Infancy*

Physiology; Ferments.—Zwiefel, 1874, and Korowin, 1875, were unable to find a diastatic ferment in the pancreas of the newborn, and to this fact is due most of the misconceptions concerning the power of digesting starch in infancy. The work which corrected this impression will be quoted later.

Saliva.—Zwiefel found diastase in the parotid gland of the newly born but was unable to find it in the submaxillary. Ibrahim, after a prolonged piece of work, found it in both the parotid and submaxillary glands, its action being stronger in the former than in the latter. Diastase was found much earlier in fetal life in the parotid than in the submaxillary, traces being found in the former at the fourth and in the latter at the sixth month of fetal life. The diastase of the parotid is the earliest digestive ferment found in the embryo.

A diastatic ferment can always be found in the saliva of healthy infants. The diastatic action of saliva may continue in the stomach as long as two hours after feeding.

Stomach.—Ibrahim is the only worker who has examined the gastric mucous membrane of the newborn for the carbohydrate splitting ferments, and he has been unable to find either lactase, maltase or invertin.

Pancreas.—Moro was able to demonstrate the presence of amylolytic ferment in the pancreas of newly born babies when the pancreas was thoroughly extracted, and thus disproved the earlier work of Zwiefel and Korowin. Ibrahim never failed to get the ferment in a six

months' fetus when he tested the action of the ferment on starch meal. He was, however, unable to find it when he tested soluble (*i. e.,* cooked) starch.

Ibrahim was unable to demonstrate invertin and lactase in the pancreas of the newborn or older babies, but he was usually able to demonstrate maltase in the newborn and always in older children. Maltase may also be found in the blood.

Small Intestine.—The mucous membrane of the small intestine contains amylolytic ferments.

Lactase, the ferment which splits milk sugar, has been repeatedly found in the mucous membrane of the small intestine. Ibrahim always found it in the small intestine and meconium of newly born babies, but was unable to find it in premature infants. He says, however, that his method of determining lactase is not capable of demonstrating small amounts. Lactase is more abundant in the young animal than in the adult.

Pautz and Vogel found maltase, the ferment which splits malt sugar, in the small intestine of infants.

Invertin, the ferment which splits cane sugar, was found in the secretions of the small intestine of the newborn by Miura, and Ibrahim was always able to demonstrate its presence both in the intestinal mucous membrane and in the intestinal contents of all fetuses.

Large Intestine.—It is difficult to wash the large intestine free from meconium, and the results of the examinations of its mucous membrane are variable, as the tables of Miura, Pautz and Vogel show. It is, therefore, impossible to say, whether it contains ferments or not.

Stools.—Pottevin found an amylolytic ferment in the meconium. Kerley, Mason and Craig were able to demonstrate the presence of a strong amylolytic ferment in the stools of very young babies, the possibility of the bacterial fermentation of starch being excluded. There is a larger amount of diastase in the stools of breast-fed babies than in those of the bottle-fed, which Hecht believes to be due to the fact that the intestinal contents of the breast-fed baby pass more quickly through the intestinal canal than do those of the bottle-fed baby.

The power of digesting starch, while occasionally absent, is, therefore, almost always present both in the fetus and in the newly born. Hedenius' experiments show that it is less powerful in young babies than in later life. Young babies are nevertheless able to adapt themselves to a food rich in carbohydrates. There is, according to Moro, a rapid increase in the power of digesting starch during the first week of life. The baby, therefore, has a power of digesting starch at birth which gradually increases in strength as the baby grows older. Ac-

cording to Finizio, it is twice as strong at eight months as it is at birth, while at twelve months it is almost as strong as at three years.

The question whether the carbohydrate-splitting ferments are affected by disease has been answered only in part. Orban found by animal experimentation that an injured intestinal mucous membrane contained no lactase, and that the stools of babies ill with enteritis contained no lactase. Langstein and Steinitz, on the other hand, always found lactase in the stools of babies ill with enteritis, whether mild or severe, acute or chronic.

Forms of Carbohydrates.—The carbohydrates used in infant feeding may be divided into the following groups:

Milk Sugar Group	Cane Sugar Group	Malt Sugar Group	
		Starch (amylum) | Dextrin (amylo- dextrin) | Erythro- and Achro- dextrin $\updownarrow$	Poly- saccharide
Lactose (milk sugar) $\updownarrow$	Saccharose (cane sugar) $\updownarrow$	Maltose (malt sugar) $\updownarrow$	Di- saccharide
Dextrose + Galactose	Dextrose + Levulose	Dextrose + Dextrose	Mono- saccharide

Digestion of Carbohydrates.—The carbohydrates are broken down during digestion into the simplest forms of sugar, the monosaccharides, by the various ferments described above. According to Rohrmann a considerable amount of the disaccharides may pass into the intestinal mucous membrane and these be split into monosaccharides. The monosaccharides are carried by the portal vein to the liver, where they are transformed into glycogen, the only difference being that dextrose is more easily converted than levulose or galactose. The pancreas has some influence on this process, because extirpation of the pancreas in dogs results in sugar in the urine and interferes with the formation of glycogen in the liver. The liver actually has the property of forming glycogen from sugar.

The purpose of the splitting of the poly- and disaccharides into monosaccharides is to prepare them for use inside the body, because the unsplit carbohydrates are not burned up in the body but are excreted in the urine. The transformation of sugar into glycogen, which is deposited in the liver and muscles, is of great importance, because this glycogen can be broken down again into sugar according to the needs of the body. The monosaccharides are absorbed more quickly than the disaccharides.

A large part of the digestion and absorption of the carbohydrates takes place in the upper part of the small intestine, but splitting and absorption may also take place in the large intestine.

There is normally about 1-10 per cent of dextrose in the blood. The slightest disturbance of the regulating apparatus will cause a hyperglycemia which results in glycosuria. A deficit of sugar in the blood is made up from the glycogen deposits.

Albertoni and Hedon found that sugars have a purgative action when they are given in large enough amounts. This action is more marked when they are taken in concentrated solution. All sugars have this action, the difference between them being only in degree. They found that glucose and cane sugar are much more quickly absorbed than lactose, and that the former has less of a purgative action than the latter.

Little or no sugar can be found in the stools under normal conditions, but when the food passes rapidly through the intestinal canal, as it does when peristalsis is rapid as the result of disease or indigestion, sugar can be found in the stool (Hecht). Usually only the products of the decomposition of sugar can be isolated.

. Hedenius fed babies with milk mixed with wheat flour, oat gruel or Keller's malt extract and measured the amount of carbohydrates ingested, the amount in the stools, and their acidity. He found that when simple cereals were used, less carbohydrate was found in the stools than with complicated mixtures and that the more carbohydrate in the stool, the greater its acidity. He never found more than 3 per cent of the ingested carbohydrate in the stool in any instance. Keller has shown that carbohydrates make the digestion of protein more complete.

Metabolism of Carbohydrates.—Numerous observations have shown that when milk sugar is injected directly into the circulation it may be completely recovered in the urine. Grosz was never able to detect milk sugar in the urine of healthy babies, but found it in the urine of those suffering from gastrointestinal disease, in which there was presumably an absence of lactase in the intestine. Langstein and Steinitz repeated Grosz's experiments and in certain instances found lactase in the stools at the same time that sugar was being excreted in the urine. This sugar was, moreover, not always lactose, but sometimes galactose, one of the products of the splitting of lactose. They tried to explain this as follows: that some of the sugar passes through functional or anatomic lesions of the intestinal wall before it is completely broken up and is excreted in the urine as an intermediary product of metabolism.

Mendel and Kelimer have shown that when cane sugar is introduced subcutaneously into dogs or cats in doses of 1 to 2 grams per kilogram of body weight, it is not completely recovered in the urine. The quantity excreted amounts, as a rule, to more than 65 per cent of that introduced. The excretion begins within a few minutes and is usually completed within thirty-six hours. Fisher and Moore draw attention to the possibility that the sugar thus introduced may be excreted through the walls of the alimentary tract and there be digested. These views are supported by Japelli and D'Errico, who conclude from their experiments on dogs that when cane sugar is introduced directly into the circulation the quantity eliminated in the urine is never equivalent to the amount injected. This causes both glycosuria and saccharosuria, the former disappearing first. The blood has no power of converting cane sugar. According to these writers, cane sugar introduced intravenously is eliminated into the alimentary tract through the gastric mucosa, the salivary glands and, to an insignificant degree, through the bile. The subsequent fate of this component is obvious.

According to Finkelstein, sugars may cause fever. This so-called sugar fever has been studied especially by his pupils. Leopold found that 43 per cent of the babies tested with lactose, 47 per cent of those with glucose, 42 per cent of those with saccharose (cane sugar), and 33 per cent of those with maltose, reacted with fever. This fever was always accompanied by diarrhea, and in none of the cases tested in which the stools remained normal did the sugar cause fever.

The limits of assimilation of the different sugars vary and are as follows:

Grape Sugar.—In babies, about 5 grams per kilogram (Langstein and Meyer).

Grape Sugar.—In one-month baby, 8.6 grams per kilogram (Greenfield).

Galactose.—No accurate data.

Levulose.—Lower for babies than adults. One gram per kilogram (Keller).

Maltose.—Over 7.7 grams per kilogram (Reuss).

Lactose.—3.1 to 3.6 grams per kilogram (Grosz).

Cane Sugar.—Probably about the same as lactose (Reuss).

Escherich divides the digestive disturbances of infancy into two main types: (*a*) Fermentation with the formation of acid products, and (*b*) putrefaction with the formation of alkalies. These two processes are antagonistic to one another, the basis of fermentation being carbohydrates and, of putrefaction, protein. An excessive preponderance of one over the other may do harm. Fermentation results in an excessive formation

of acids, especially of lactic acid from lactose. This may cause a large number of stools as the result of the increased peristalsis and the flow of serous fluid or cellular exudate. Razenski has shown that in babies sick with what he calls "dyspepsia intestinalis acida lactorum" there is an increased acidity of the intestinal contents and that the utilization of fat is diminished. Meyer and Leopold intimate that a sugar indigestion may cause the appearance of casein curds.

Finkelstein and Meyer believe that milk sugar is the primary cause of the fermentative dyspepsias of infancy and that when there is a disturbance of the utilization of fat in these conditions it is a secondary manifestation. They claim that these dyspepsias can be relieved by the long-continued diminution of the carbohydrates in the food and quickly cured by the withdrawal of milk sugar and the administration of large amounts of casein (*Eiweissmilch*). In general, the fermentation of the sugar depends upon the relation between the casein and the sugar in the food. They advise the administration of other easily assimilable and consequently little fermentable carbohydrates, such as maltose, in place of lactose, after the disappearance of the acute symptoms. Birk and Reuss and Sperk have confirmed their observations. Braumüller called attention, however, to the danger of the sudden addition of sugar to the diet after it has been withdrawn and large amounts of casein given, believing that under these conditions the ability to form the ferments necessary to take care of sugar is practically abolished.

Kendall has shown that the colon bacillus, like the diphtheria and tetanus bacilli, causes fermentation or putrefaction according to whether it is in a carbohydrate or protein medium, that it attacks the sugar in preference to the protein of the medium and that until the carbohydrate is used up the protein is shielded from attack. The products of the fermentation of carbohydrates are acid. He also showed that the Shiga bacillus produces toxin only when the medium has an alkaline reaction. He, therefore, proposed feeding babies ill with bacillary dysentery with an easily fermentable carbohydrate in order to change the character of the bacterial activity in the alimentary canal from the proteolytic to the fermentative type. The result is to stop further formation of toxin. This was done by feeding the babies a 5 per cent lactose solution, under the influence of which the dysentery bacillus and streptococci tend to disappear.

Fat Metabolism

The infant obtains its required amount of fat elements in the breast or bottle milk. But whereas the infantile system assimilates almost 96 per cent of the fat of breast milk, it absorbs only 80 per cent of that

of cow's milk. Furthermore, the daily loss of fat by infants fed on mother's milk is only half as great as that of cow's milk. As in adults so in children fat plays an important rôle in the maintenance of body heat and, if combined with proteids, it saves nitrogenous waste and to a certain extent replaces the carbohydrates.

The fats of the milk pass unchanged into the duodenum. Here they are partly emulsified and in part split up by the pancreatic juice into fatty acids and glycerine, and thus largely absorbed by the intestinal villi. The unassimilated fat passes out with the feces as neutral fats, fatty acids and soap.

L. E. Holt, A. M. Courtney and H. L. Fales, have made a very exhaustive study of the "Fat Metabolism of Infants and Young Children" (Am. Jour. Dis. Child. Vol. xvii and xviii, 1919) and arrived at the following conclusions regarding

FAT RETENTION AND EXCRETION IN
RELATION TO DIET

I. Fat in the Stools of Breast Fed Infants

1. The fat of the stools of normal breast fed infants, according to their observations, averaged 34.5 per cent of the dried weight and frequently was as high as 50 per cent.

2. The soap fat in the best stools predominated over the other forms of fat, averaging 57.8 per cent of the total fat, as determined on the dried stool. The average stool of the normal breast fed infants showed a soap fat of 43.1 per cent of the total fat, as determined on the dried stool, which would correspond to over one-third of the total fat of the fresh stool.

3. The neutral fat in the best stools averaged 15.9 per cent of the total fat; in the average stool the neutral fat was 20.2 per cent of the total fat. The amount of neutral fat is not affected by the drying process.

4. No constant relation was shown between the per cent of fat in the mother's milk and the per cent of total fat and its distribution in the stool.

5. With a higher total intake of fat, the fat per cent and the soap fat in the stool were somewhat increased.

6. A range of fat absorption from 90.3 to 99.2 per cent of the intake was found in healthy breast fed infants.

II. Fat in the Stools of Infants Fed on Modifications of Cow's Milk

1. The material presented in this article comprised the results of analysis of 128 stools of seventy-seven infants whose ages ranged from 2 to 18 months, fed on modifications of cow's milk.

2. The average fat per cent of the dried weight in normal stools was 36.2. The hard, constipated stools showed no variation from this figure. In the stools not quite normal in appearance the average fat per cent was slightly lower. In severe diarrhea the fat per cent of dried weight was much higher, reaching an average of 40.7 per cent.

3. The soap per cent of total fat was very high in both normal and constipated stools, averaging, respectively, 72.8 and 73.8 per cent. As the stools became less normal in appearance the soap fat diminished rapidly and averaged in the loose stools only 30.6 per cent of the total fat, in the diarrheal stools 12.4 per cent, and in those of severe diarrhea only 8.8 per cent of the total fat.

4. The neutral fat was less than 10 per cent of the total fat in normal and constipated stools. It increased as the soap fat diminished and in diarrheal conditions made up about 60 per cent of the total fat in the stool.

5. The free fatty acids constituted about 17 per cent of the total fat of normal and of constipated stools. It was increased somewhat as the stools became less like the normal and in the diarrheal stools was over 30 per cent of the total fat of the stool.

6. No definite relationship was shown between the daily fat intake and the per cent of fat or the distribution of fat in the stool.

7. The average per cent of the fat retained with normal stools was 91.3 per cent of the intake. The retention was but little lower when the stools were somewhat harder or softer than normal, or were not homogeneous, or contained more or less mucus without being distinctly watery. As the water in the stools increased, the per cent of retention dropped markedly, reaching in severe diarrhea 58.4 per cent of the intake.

8. There was no striking relation between the fat intake and the per cent of the intake retained, except when the intake was abnormally low.

III. Fat in the Stools of Children on a Mixed Diet

1. In the normal or constipated stools of older children whose diet consisted of milk alone or milk with bread and cereal the fat percentage of dried weight averaged 30.7, which is lower than the average

found for similar stools of infants taking modifications of cow's milk. The soap percentage of total fat averaged 60.9, which was somewhat lower than that found in the stools of the infants.

2. The normal and the constipated stools of children on a mixed diet showed almost identical average values both for fat percentage of dried weight and for distribution of fat. The fat percentage of dried weight averaged, respectively, 18.0 and 20.1, and the soap averaged, respectively, 45.1 and 47.9 per cent of the total fat. These values were much lower than those found when the diet contained little or no solid food.

3. In the acid abnormal stools of children on a mixed diet the fat averaged 15.1 per cent of the dried weight. Both the fat percentage of dried weight and the soap percentage of total fat were much lower than in normal stools and the values for fatty acids and for neutral fat were higher.

4. With rachitic children the fat percentage of dried weight averaged 34.7 in the alkaline stools, and 24.6 in the acid stools.. The values were higher than those found for corresponding types of stools of normal children. The proportions of soap, fatty acids and neutral fat were not significantly different from those for normal children.

5. The stools of children suffering from chronic intestinal indigestion showed a much higher fat percentage of dried weight than those of normal children; the average for alkaline stools being 36.4 per cent, and for acid stools 35.3 per cent. The average percentage of neutral fat was lower in both alkaline and acid stools of these children than in the stools of normal children. The fatty acids were higher than normal, much higher when the reaction of the stools was acid.

6. The average fat loss in the stools of normal children varied between 2.6 and 3.0 gm. in all the groups studied, being highest in the stools of children whose diet contained the smallest proportion of solid food and the largest proportion of milk.

7. The normal children on mixed diet retained on the average about 94 per cent of the fat intake, regardless of the type of stool. The average actual retention was about 38 gm. daily. The children with little or no solid food and a smaller fat intake showed a lower actual, and a somewhat lower percentage retention than those on a general mixed diet.

8. The rachitic children showed a slightly larger fat loss in the stools than did the normal children; their intake, however, was higher. Their actual retention, therefore, equalled or exceeded that of the normal children, and their percentage retention was only a little lower than the normal average.

9. The fat loss in the stools of the children suffering from chronic intestinal indigestion was very great, averaging 7.3 gm. daily in the alkaline stools and 8.0 gm. in the acid stools. Both the actual and percentage retention were much lower than normal. The percentage of the intake retained averaged 79.1 when the stools were alkaline and 77.7 when they were acid. When the intake of fat was very high the actual retention was usually as high as that found for normal children.

IV. The Digestion of Some Vegetable Fats by Children on a Mixed Diet

1. The stools of children receiving a considerable proportion of vegetable fat did not differ essentially in appearance from those of children receiving mainly milk fat, although they were usually somewhat softer.

2. The fat percentage of dried weight of the stools averaged somewhat lower when nut butter was taken, and somewhat higher when corn oil was taken, than when the fat in the diet was mainly milk fat; and when large quantities of corn oil were included in the diet the average was much higher.

3. The soap percentage of total fat in the stools was usually a little lower and the neutral fat a little higher with vegetable fat than when the fat of the diet was mainly milk fat.

4. When nut butter was taken the fat excretion in the alkaline stools was lower and in the acid stools it was higher, than when the diet did not contain vegetable fat. When corn oil was taken in considerable amounts the fat excretion in the stools was higher than when the fat of the diet was mainly milk fat. However, the total fat intake when corn oil was included in the diet was very large and the actual retention of fat always much higher than the normal average for mixed diet. When vegetable fat formed a considerable part of the total fat intake, the percentage of the fat intake retained was usually higher than the normal average. In a few instances when the stools were acid and in a few when large amounts of corn oil were taken, the percentage retained was low.

5. The individual children observed for considerable periods with changes in the kind and amount of fat intake showed quite as good digestion of vegetable fat as of corresponding amounts of milk fat and no unfavorable effect on general health and nutrition was observed. No children were kept long enough on a diet presumably deficient in fat-soluble A to warrant any conclusions as to the effect of such a diet upon growth and health. In the case of one child who for

five weeks was on a diet in which there was no definite source of fat-soluble vitamin, 95 per cent of the fat of the diet being corn oil, he ceased to gain in weight, but showed no loss and the general health continued excellent. The fact may not be without significance that of six children, 80 to 95 per cent of whose fat intake was vegetable fat, two developed styes and two others eczema upon the face, which disappeared when the diet was changed to include milk fat.

Woman's Milk Feeding

Woman's milk* is a highly nutritious, biologically as yet somewhat mysterious product, destined by nature to serve as the food
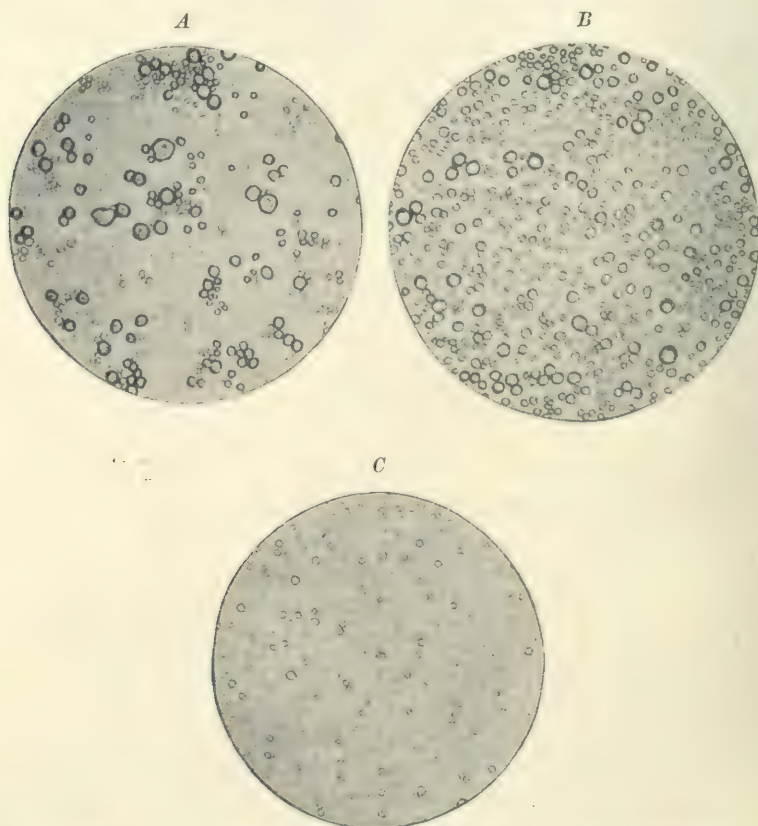
Fig. 1.—Microscopic appearances of woman's milk. (After *Fleischman.*) *A.* Poor milk showing preponderance of large fat globules and a paucity of fat. *B.* Normal milk, showing the preponderance of medium-sized fat globules. *C.* Poor milk; a paucity of fat and an almost granular state of the fat globules.

*For its Approximate Composition see footnote, p. 48.

PLATE I
THE NORMAL STOOL OF THE BREAST-FED INFANT
(Courtesy Dr. Hector Charles Cameron.)

supply alike for the rich and the poor, the weak and the strong infant under nine months of age. It not only complies with the aforementioned qualitative requisites, but being ready for immediate consumption—without previous pasteurization, sterilization or modification—at all hours of the day and at all seasons of the year, it is also the most convenient and satisfactory food from an economic point of view. Infants reared on woman's milk are almost invariably healthier, stronger and less troublesome than those that are bottle fed. With suitable management they are, as a rule, free from gastroenteric affections, scurvy and rickets, and present greater power of resistance to communicable diseases.

Maternal Nursing.—For the reasons just given, and in view of the facts that wet-nurses are expensive luxuries, are often unreliable, and may at some time during the nursing period, through unscrupulous and impure contact, contract and convey a disease to her charge, it is the solemn duty of every healthy mother to endeavor to nurse her offspring, wholly or partially, even if it be only for a brief period of time.

Successful maternal nursing presupposes, in addition to general good health of the mother, well-developed breasts and nipples and an ample supply of milk. These qualifications are rarely met to perfection in women of large cities, where the extravagances of extreme wealth or the misery of extreme poverty sap their vital forces. A great deal, however, can be accomplished by judicious management of the mother during pregnancy and parturition.

The prospective mother should be placed in the most healthful physical and mental condition. Her diet should be liberal, her living rooms spacious and airy and her surroundings cheerful. She is to be free from anxieties of a livelihood and the pompous frivolities of wanton society. The primipara should be taught to realize that pregnancy and parturition are physiologic processes, ordinarily devoid of perilous complications or sequelæ.

Toward the end of pregnancy the breast nipples should be elongated by gentle traction with the fingers or pump, and cleansed and hardened by means of hot boric acid solutions, cognac, glycerite of tannin, and the like. To insure an ample supply of breast milk after delivery, in addition to complying with the aforementioned suggestions, a liberal fluid diet, consisting principally of rich cow's milk, cornmeal and oatmeal gruel cooked in milk, malted milk, etc., forms the most efficient adjuvant. At a later period the dietary of the nursing mother should be increased by a liberal allowance of meat, eggs, vegetables and other nutritious foodstuffs to which she was ordinarily accustomed.

Light outdoor exercise, regulation of the bowels, avoidance of fatigue and nerve disturbances, all serve well to improve the health of the mother and the quality of her milk and indirectly to promote the welfare of the baby.

One other special advantage of maternal- over wet-nursing is the benefit the newborn derives from the consumption of the provisional milk secretion—*the colostrum*. This deep yellow, strongly alkaline and albuminous fluid which forms the mammary secretion during the first three or four days after labor, not only acts as a laxative—which is badly needed, but being small in quantity it also serves to moderate the greedy appetite of the infant and prevents early overfeeding, the usual cause of infantile colic.

The nursing of the baby is generally begun about eight hours after delivery, or later if the mother has not fully recovered from the painful and fatiguing ordeal. During the first few days the infant is
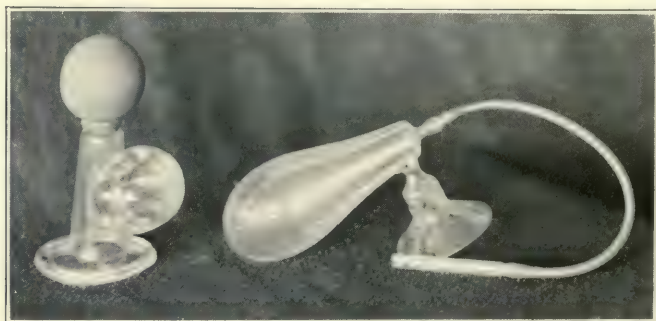
Fig. 2.—Breast pumps.

applied to the breast every three or four hours and afterwards every two and a half or three hours. It should not be awakened for a feeding if sound asleep (except when very weak and delicate), and, unless very restless, should be left alone from 10 p. m. to 5 a. m. It should be nursed from fifteen to twenty minutes at a time, alternately on one and the other breast, or on both breasts if the milk secretion is scanty. From six weeks on the infant should be fed every three hours, and less frequently when it reaches six months of age. Between nursings the baby may receive a few ounces of warm water.

Before and after each feeding the breast nipples should be carefully cleansed with a warm saturated solution of boric acid.

If the breast nipples are short, sunken or cracked, we must temporarily resort to an artificial nipple or breast pump (Fig. 2). The latter device is also employed where the infant is too weak to pull, or refuses to make an effort to do so. In very delicate infants, *e. g.*, pre-

matures, it is often necessary to withdraw the breast milk with a pump and to administer it by means of a spoon or dropper.

With the suggestions here offered the majority of healthy mothers will be able to nurse their offspring, provided they are sufficiently encouraged by the physician and the enormous advantages of maternal breast feeding are thoroughly explained to them.

When an infant does not thrive on breast milk, it is imperative, before resorting to another infant food, to analyze carefully the breast milk, and, if possible, to overcome the difficulty. We should determine:

1. *The Quantity*—This can readily be learned by extracting the milk supply of one or both breasts, or by weighing the infant before and after
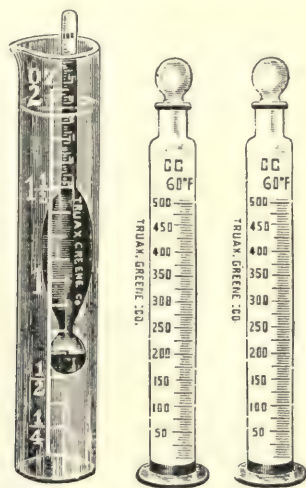
Fig. 3.—Holt's milk testing apparatus.

nursing and noting the difference in weight—the gain in ounces indicating the amount of milk it has obtained.

2. *The Quality.*—As the sugar is usually found to be normal in all cases, the tests are ordinarily limited to the fat and protein contents of the breast milk. After obtaining an ounce of what is called ''middle-milk'' (*i. e.*, the milk collected after 1 or 2 ounces had been withdrawn) or of the entire breast supply, we determine the following qualifications:

(*a*) *Reaction.*—Fresh breast milk should be alkaline or neutral and may be tested with litmus paper.

(*b*) *Specific Gravity.*—This should be about 1.030, taken by means of a lactometer, at a temperature of 65° to 72° F.

(*c*) *Fat Content.*—The cylinder of Holt's milk set (Fig. 3) is filled with the sample of breast milk up to the zero mark and allowed to

stand for twenty-four hours in a room temperature of 70° F. The percentage of cream is then read off, bearing in mind that the ratio of the cream to the fat is approximately 5 to 3, *i. e.,* 5 per cent of cream equals 3 per cent of fat.

(*d*) *Proteids.*—The amount of proteids is approximately determined by the amount of fat and the specific gravity of the milk, *i. e.,* high specific gravity, high proteids; low specific gravity, high fat. Holt's accompanying table explains the application of this principle:

	SPECIFIC GRAVITY	CREAM (24 HOURS)	PROTEID (CALCULATED)
Average	1.031	7	1.5%
Normal variations	1.028-1.029	8%-12%	Normal (rich milk)
Normal variations	1.032	5%-6%	Normal (fair milk)
Abnormal variations	Low (below 1.028)	High (above 10%)	Normal or slightly below
Abnormal variations	Low (below 1.028)	Low (below 5%)	Very low (very poor milk)
Abnormal variations	High (above 1.032)	High	Very high (very rich milk)
Abnormal variations	High (above 1.032)	Low	Normal (or nearly so)

While, as a rule, the breast milk of the modern mother is characteristic for its paucity, we occasionally come across breast milk that is too rich in quality, especially as regards the fat content. In the majority of such instances, if the excess in fat is detected early, it can readily be corrected (by reducing the mother's diet, encouragement of active exercise, etc., or by resorting to partial nursing) before any appreciable harm has been done to the infant. In some cases, however, the abnormality of the milk is not discovered until the infant is suffering from "fat indigestion" (diarrhea with curds of fat, eructations, colic and possibly loss of weight), and one is often in a quandary as to what is best to do. An attempt may be made to thin the breast milk by administering to the infant before each nursing ½ or 1 ounce of plain or cereal water. Or the baby is allowed to nurse at the breast only a few minutes and is then given an ounce or two of diluted skimmed milk to make up the quantity to satisfy the baby. If these procedures and the dieting of the mother fail, and the child is progressively getting worse, we must either engage a wet-nurse or put the baby on a suitable artificial food.

Where the milk supply is deficient, partial nursing should be insisted upon, preferably alternating one breast- with one bottle-feeding.

Wet-Nursing.—Wet-nurses at best are an evil, but often indispensable, where mothers will not, cannot, or must not nurse their own offspring. If the mother cannot nurse her baby because of quantitative or qualitative insufficiency of her milk, there is no urgency of securing

a wet-nurse, as the milk may be improved by a richer diet and better care of the mother, or the infant may receive daily two or three feedings of properly modified cow's milk. In the event, however, that the mother is utterly unable to nurse her baby or is prevented from doing it through disease (tuberculosis, cancer; acute, greatly debilitating affections; advanced kidney or heart disease; local inflammation of the breast, psychoses and the like) or pregnancy, a wet-nurse is the best substitute. The wet-nurse to be chosen must undergo a very careful physical examination, first as to the secreting quality of the breasts and the condition of the nipples, and next as to her general health.

The secreting quality of the breast is best tested by "stripping," *i.e.*, by grasping the upper portion of the *nipple* with the thumb and two fingers, and, while moving the fingers briskly forward, exerting uniform but gentle pressure. With this manipulation the milk should escape from the breast in several even jets for from fifteen to thirty seconds. Too much reliance should not be placed upon the form of the breast, for even pendulous, cylindrical, or conical breasts are occasionally poor milk producers. On the other hand, an abundance of glandular parenchyma offers more reliable guarantee as to its secreting power. The physician should be on his guard that the abundance of milk be not the result of the breast having been allowed to fill up for several hours previous to the examination—a fact recognized by the presence of pain on pressure and intense distention of the mammary ducts. The nipples should be hard, long and bulky, free from *severe* ercsions or fissures.

The quality of the milk is not nearly as essential as the quantity, since the former can usually be improved upon by suitable diet and good hygiene.

The following diseases render a wet-nurse useless: Tuberculosis, whether local or general; syphilis, in all its stages (not necessarily contraindicated in the mother); noncompensating heart disease; grave affections of all other bodily organs; profound anemia; intractable, communicable skin, hair, and eye diseases; gonorrhea; suppurative processes of the bones; mastitis (not necessarily contraindicated in the mother); ozena, drug addiction, psychoses, and epilepsy.

The possible presence of syphilis should receive special attention. Corona veneris, bony tumefactions, nasopharyngeal patches, old ulcers and scars, enlarged glands (especially paramammary, epitrochlear, and inguinal) should invariably arouse the suspicion of the examiner. Every wet-nurse should undergo a Wassermann test.

The wet-nurse of choice should be one between twenty and thirty years, who has given birth to two healthy children and nursed one suc-

cessfully, the age of the last child being nearly the same as the one she is about to nurse. The diet of the wet-nurse, the care of her breasts and nipples, the mode of living, exercise, etc., should be the same as in a nursing mother (q. v.). Sudden changes, however, from dire want to superabundance are to be avoided.

Artificial Feeding

Where maternal nursing is impossible, and wet-nursing impracticable, there is nothing else left but to resort to artificial feeding. All human ingenuity and skill have thus far failed to provide a food for infants that is as nutritious, digestible, sterile without interference of composition, and as economic as woman's milk.

Cow's Milk Feeding

With suitable modification cow's milk forms the best substitute for human milk. But it is a poor substitute at best, for not alone does human milk vastly differ from cow's milk in the quantitative proportion of the essential chemical ingredients, but the latter vary greatly also qualitatively. Furthermore, human milk contains several as yet not fully determined biologic constituents, especially enzymes, which are absent in cow's milk.

To meet the aforementioned requirements of an infant food, cow's milk must undergo considerable modification to approach human milk in its composition. As may be noted from the comparative table* of human and cow's milk, the latter contains about five times as much of casein and only half as much of lactalbumin as the former. Cow's milk-casein coagulates in the stomach in large firm curds which dissolve slowly, the opposite being the case with human milk-casein. Human milk-fat forms a finer emulsion than cow's milk-fat, and contains a much smaller amount of fatty acids. The salt in human milk is richer in iron and, finally, human milk is sterile, whereas cow's milk when reaching the consumer is replete with bacteria.

To equalize the numerous differences between the two milks, so as to render cow's milk both digestible and nutritious, we have to proceed as follows:

1. Reduce the quantity and coagulability of the casein by:

---

*Approximate Composition of Human and Cow's Milk:

	HUMAN	Cow's
Water	87	88
Solids	13	12
Caloric value	750	. 650
Lactalbumin and globulin	0.75 to 1	0.3 to 0.5
Casein (combined with calcium)	0.5 to 0.75	3.0
Fat	4.0 to 4.5	3.5 to 4.0
Milk sugar	7.0	4.0 to 4.5
Salts	0.20	0.7

(*a*) Dilution; adding, for example, to 1 ounce of cow's milk 2 ounces of either plain, or cereal water, a milk mixture is obtained containing but 1 per cent of protein, which can readily be assimilated by a young infant. Although it has recently been claimed that normal babies can tolerate large amounts of cow's milk-casein, we must be cautious to accept these views as final, since the medical profession has lately been swayed too often from one extreme view to the other. Lime water is usually added, by its alkalinity, to delay the coagulation of the casein; and cereal diluents act both as a food and protective colloids which hinder the formation of large casein curds. Starches in large quantities, however, are not very readily digested by infants under two or three months of age.

(*b*) Addition of sodium citrate, 1 to 2 grains to every ounce of milk or cream. Sodium citrate acts by combining with calcium caseinate of the milk to form calcium citrate and sodium caseinate, which, after being split up by the rennin, changes into soluble sodium paracaseinate.

(*c*) Boiling the milk for five minutes in a single boiler.

(*d*) Peptonization (p. 59).

2. Increase the sugar (which has become reduced in quantity by dilution) by the addition of either milk sugar, malt sugar or cane sugar, the quantity varying with the degree of dilution of the milk mixture. Ordinarily one-half of a teaspoonful for each ounce of the diluent* will be required. There is still considerable divergence of opinion as to which of disaccharides are best suited for infant feeding. I am inclined to think that milk sugar probably serves best for normal babies, while maltose or dextrimaltose is best in digestive disturbances. Cane sugar is more useful in constipation.

3. Augment the fat content, if diminished by dilution of the milk, by the addition of gravity cream (the cream that is visible on bottle milk after setting for six hours or longer) or preferably by using "top milk"†

*Mode of Preparation of Diluents for Cow's Milk.—*Barley water.*—One tablespoonful of prepared barley (Robinson's) is rubbed up in a little cold water; to this is gradually added a quart of boiling-hot water, and the mixture is allowed to boil slowly (simmer), with constant stirring, for about twenty minutes and then strained. Boiled water is then added sufficient to make one quart. *Ground groats* is especially useful in constipation.

Oatmeal water.—One tablespoonful of oatmeal is rubbed up in a little cold water; to this is added a pint of boiling-hot water and allowed to boil slowly (simmer) for one to two hours, with frequent stirring, and strained through gauze. Boiled water is then added sufficient to make one pint.

Rice water.—One tablespoonful of ground rice to a quart of water, prepared the same as barley water.

†Top milk.—Bottle milk, as obtained from reliable milk dealers, contains approximately the following percentages of fat and proteids:

PORTION TAKEN			FAT	PROTEIDS
Upper ½ ounce			24.8	3.1
"	1	"	23.1	3.2
"	2	"	21.4	3.3
"	4	"	20.1	3.4
"	6	"	18.6	3.5
"	8	"	16.7	3.6
"	12	"	12.1	3.7
"	16	"	8.4	3.8
"	18	"	6.5	3.9

as a base, *i. e.*, by taking instead of "whole" milk a sufficient amount of milk of the upper 18 ounces of a bottle (which contains 6.5 per cent of fat) decanted for this purpose and thoroughly mixed (Fig. 4).

4. Insure the absence of pathogenic bacteria in cow's milk by observing the following suggestions:

The cow must be free from disease, especially from tuberculosis as determined by the tuberculin test and by regular inspection by a competent veterinary surgeon.

The cow's entire body should be groomed daily and, immediately before milking, the belly, tail, and particularly the udder should be carefully cleansed with a clean, damp cloth, with or without soap, and dried with a clean towel.

The milker must be free from communicable affections. Before milking, he should thoroughly scrub and dry his hands and don clean
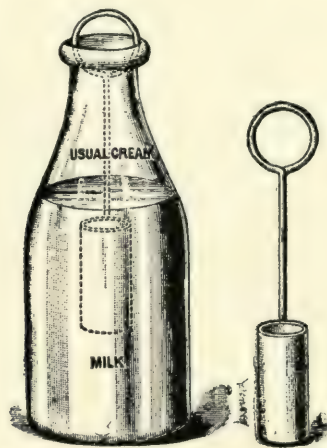
Fig. 4.—Chapin's dipper for removal of "top-milk".

washable, outer garments. He should have a few of these on hand, in order to change them should one gown or suit accidentally get soiled in the act of milking.

The milk of each cow should be collected separately in sterile utensils and immediately removed from the stable to a clean place specially reserved for the keeping of the milk until ready for shipment.

The milk should be rapidly cooled (below 45° F.) and strained through a sterile strainer, then bottled, closed with sterile discs, capped, and finally iced—all within an hour or so after milking.

Owing to the rapid development of bacteria in milk over twenty-four hours old, the milk should reach the consumer within this pe-

riod of time. The milk should further be kept on ice until needed for the preparation of the food.

If, notwithstanding all the prophylactic measures, some doubt still remains as to the sterility of the milk, we must subject it to steriliza-tion or pasteurization.*

Laboratory and Home Modification of Cow's Milk

We have just learned the numerous essential differences of com-position that exist between human and cow's milk, and the means by which the differences can be removed. Were it merely a question of obtaining milk of a definite uniform composition which would at once prove suitable for the feeding of infants of all ages, the problem of artificial feeding of infants would long have been solved. Unfor-tunately this is not the case. Not only must cow's milk be modified so that its principal constituents greatly resemble those of human milk, but it must undergo also specific modification to meet the digestive powers and the requirements of the individual infant at certain periods of life—quite a difficult proposition indeed.

Thanks to the rapid strides of physiologic chemistry and the good will and enterprise of several milk dealers and laboratory chemists, the modification of cow's milk as an infant food has almost reached a stage of perfection. With the help of the laboratory chemist, the physician is now enabled to write a prescription for a food mixture of definite composition and, like a drug in the pharmacy, have it com-pounded exactly as ordered. The latitude of composition is well illus-trated in the prescription form on page 52.†

When "laboratory milk" is not obtainable, and "home modifica-tion" has to be resorted to, we may greatly facilitate the process and obviate the difficult task of memorizing complicated formulas by se-

---

*Sterilization and Pasteurization.—Both of these processes are accomplished by means of one of the many sterilizers on the market. In *sterilizing,* the milk is heated for about fifteen minutes at a temperature of 212° F.; in *pasteurizing,* for about forty minutes at a temperature of from 140° to 150° F. For infant-feeding the milk should undergo the heating process after it has been modified and divided in the requisite number of feeding bottles for the entire twenty-four hours. The bottles are cooled off by allowing cold water slowly to run through the sterilizer; they are then tightly corked, preferably with nonabsorbent cotton, and placed on ice until needed for use. Before feeding the bottle should be warmed to body heat. Except during the hot summer months or when there is good reason to believe that the milk harbors virulent bacteria (*e. g.,* during epidemics of typhoid, cholera, etc.), sterilization is nowadays rarely practiced. Pasteurization is usually resorted to instead, particularly since it has been demonstrated that this process is less apt to change the taste of the milk, to interfere with its digestibility, or to cause constipation. The view held, especially by overenthusiastic, though well-meaning, laymen, that pasteurized milk is as nutritious as clean, fresh, raw cow's milk, is not based upon scientific observation. Quite the contrary; pasteurized milk lacks several nutritive and protective elements that exist in fresh cow's milk. Hence, its continued use greatly interferes with the growth and development of the infant, and is not rarely productive of rickets and scurvy, if the baby is fed on milk exclusively.

†From Morse and Talbot's "Diseases of Nutrition."

PER CENT

Fats.. .....

(a) Carbohydrates.................... { Lactose (milk sugar)
Maltose (malt sugar)
Sucrose (cane sugar)
Dextrose (grape sugar)
Starch...................

(b) Dextrinize...

(c) Proteins { Whey...
Casein...

(d) Peptonize...

(e) Sodium citrate { % of milk and cream.....................
% of total mixture........................

(f) Sodium bicarb { % of milk and cream........................
% of total mixture........................

(g) Lime water { % of milk and cream.......................
% of total mixture........................

(h) Lactic acid bacillus { 1. To inhibit the saprophytes of fermentation_.
2. To facilitate digestion of the proteins......

Heat at......° F.   Number of feedings......   Amount of each feeding.........

Ordered for...

Date.......................   ..................................M.D.

EXPLANATORY

(a) It requires 0.75 per cent starch to make the precipitated casein finer.

(b) One hour completely dextrinizes the starch.

(c) In case physicians do not wish to subdivide the proteins, the words "whey" and "casein" may be erased.

(d) Twenty minutes render the mixture decidedly bitter.

(e) It requires 0.29 per cent of the milk and cream used in modifying to facilitate the digestion of the proteins; i.e., the formation of a soft curd; 0.40 per cent to prevent the action of rennet; i.e., the formation of tough curds.

(f) It requires 0:68 per cent of the milk and cream used in modifying to favor the digestion of the proteins; 1.70 per cent of the amount of milk and cream used suspends all action on the proteins in the stomach; 0.17 per cent of the total mixture gives a mild alkaline food.

(g) It requires 20 per cent of the milk and cream used in modifying to favor the digestion of the proteins; 50 per cent of the amount of milk and cream used suspends all action on the proteins in the stomach. Five per cent of the total mixture gives a mild alkaline food.

(h) Percentage figures represent the per cent of lactic acid attained when the food is removed from the thermostat. When the lactic acid bacillus is used to facilitate the digestion of the proteins, this is the final acidity, as the process is stopped by heat at this point. When the bacillus is used to inhibit the growth of saprophytes the acidity may subsequently increase to a variable degree, as the bacilli are left alive; 0.25 per cent lactic acid just curdles milk; 0.50 per cent gives thick curdled milk; 0.75 per cent separates into curds and whey.

lecting a "standard" milk formula of simplest composition (1:1, i. e., 1 ounce or its multiple of milk to 1 ounce or its multiple of a diluent) and preparing the other milk mixtures by modifying this "standard" formula.

Directions.—1. Bear in mind the standard formula (1:1), which is intended for an infant three months old.

2. For infants *under* three months increase about every month downward the *diluent* by 1 ounce or its multiple, using "top milk" (upper 18 ounces) as a base and plain water as the diluent.

3. For infants *over* three months of age, increase every two months upward the *milk* by 1 ounce or its multiple, using "whole milk" as a base and cereal water as a diluent.

4. Add to each ounce of the diluent from $\frac{1}{3}$ to $\frac{1}{2}$ teaspoonful of sugar (milk, malt or cane sugar) and 1 teaspoonful of lime water.

1:5 one week 1:4 two weeks 1:3 one month 1:2 two months	Standard Formula milk 1:1 diluent three months	2:1 five months 3:1 seven months 4:1 nine months 5:1 eleven months

Milk modified in accordance with these suggestions yields milk mixtures of the following approximate composition:

For an infant one week old (1:5).

Top milk................... 2-2/3 ounces   Proteins 0.50
Lime water................. 3   drams   Sugar 6.00
Water......................13   ounces   Fat 1.00
Sugar...................... 4-1/3 drams
Divide in 8 bottles; give a feeding every three hours during the day and night, if the baby is awake.

For an infant two weeks old (1:4).

Top milk................... 4   ounces   Proteins 0.6
Lime water................. 1/2 ounce   Sugar 6.00
Water......................15-1/2 ounces   Fat 1.20
Sugar...................... 2/3 ounce
Divide in 8 bottles; give a feeding every three hours during the day and night, if the baby is awake.

For an infant one month old (1:2).

Top milk................... 6-1/2 ounces   Proteins 0.75
Lime water................. 3/4 ounce   Sugar 6.00
Water......................17-1/2 ounces   Fat 1.50
Sugar...................... 3/4 ounce
Divide in 7 bottles; give a feeding every three hours during the day and night, if the baby is awake.

For an infant two months old (1:3).

Top milk...................10   ounces   Proteins 1.00
Lime water................. 1-1/4 ounces   Sugar 6.00
Water......................19   ounces   Fat 2.00
Sugar...................... 7/8 ounce
Divide in 7 bottles; give a feeding every three hours during the day, and once during the night if the baby is awake.

For an infant three months old (1:1).

Whole milk	18 ounces	Proteins	1.50
Lime water	2-1/4 ounces	Sugar	6.00
Barley water	16 ounces	Fat	2.00
Sugar	2/3 ounce		

Divide in 7 bottles; give a feeding every three hours during the day, and once during the night if the baby is awake.

For an infant five months old (2:1).

Whole milk	26 ounces	Proteins	2.00
Lime water	3-1/4 ounces	Sugar	6.00
Barley water	10 ounces	Fat	2.60
Sugar	1/2 ounce		

Divide in 6 bottles; give a feeding every three hours.

For an infant seven months old (2:1).

Whole milk	32 ounces	Proteins	2.25
Lime water	4 ounces	Sugar	6.00
Barley water	7 ounces	Fat	3.00
Sugar	1/2 ounce		

Divide in 6 bottles; give a feeding every three hours.

For an infant nine months old (4:1).

Whole milk	34 ounces	Proteins	2.45
Lime water	4-1/2 ounces	Sugar	6.00
Barley water (concentrated)	4 ounces	Fat	3.25
Sugar	1/3 ounce		

Divide in 5 bottles; give a feeding every four hours.

For an infant eleven months old (5:1).

Whole milk	37-1/2 ounces	Proteins	2.50
Lime water	4-3/4 ounces	Sugar	6.00
Barley water (concentrated)	3 ounces	Fat	3.50
Sugar	1/4 ounce		

Divide in 5 bottles; give a feeding every four hours.

For infants over a year, give undiluted whole milk.

The method of home modification of milk here described, while not very exact, is based upon clinical experience, and has the further advantage over many other methods in vogue in that it does not require the knowledge of higher mathematics for its calculation. Infant feeding by calories, while very ingenious, is hardly applicable in the feeding of infants under three or even six months of age, since it provides amounts of fat or protein often entirely beyond the infantile digestive capacity. According to Heubner* an infant requires a daily ration of about 45 calories for every pound of its weight during the first quarter of a year; 40 calories during the second quarter; 35 during the third; and 30 during the fourth quarter. Fifteen grains (1 gram) of protein or carbohydrates furnish 4.1 calories and 15 grains of fat, 9.3 calories; or 1 ounce of whole milk, 20 calories. Taking for

---

*Both Heubner's and Budin's suggestions work well in breast feeding.

PLATE II

Formed Alkaline Stools

A marked example of the formed, alkaline stools, consisting largely of soaps, sometimes found in feeding with cow's milk, without excess of fat; or with "protein" milk. They are associated with increased putrefaction but diminished fermentation in the intestinal canal.

(Courtesy Dr. Hector Charles Cameron.)

example an infant one month old, ordinarily weighing 8 pounds, it would require in twenty-four hours, $8 \times 45 = 360$ calories, *i. e.,* either 18 ounces of whole milk, which would be entirely too rich in casein for an infant of that age, or 4½ ounces of gravity cream greatly diluted, which would be too rich in fat and too poor in protein and sugar. The same fault is to be found in Budin's method of giving daily an amount of milk equal to 10 per cent of the body weight of the baby.

The keynote of successful artificial feeding is individualization, *i. e.,* the selection of a food in proper proportions as to fat, sugar, and protein suitable for each individual baby's power of digestion and assimilation, and in sufficient quantities. The amount of food needed by the healthy infant is best judged by the capacity of the stomach*, subject, of course, to variations as to size, activity, etc. The question of the proportion of the food elements must be decided from time to time in each individual case, after considering the gain or loss in weight under the respective food, and watching the consistency, etc., of the bowel movements.

Indications of Faulty Assimilation of the Food

All disturbances of digestion, be they due to an excess of protein, sugar, or fat, have several symptoms in common, thus: Restlessness, flatulence, colic, loss in weight, frequent defecations and vomiting; in acute indigestion also moderate or high fever. To determine whether the digestion of fat, sugar, or protein is at fault, we have to examine the vomitus and feces.

In *fat* digestion, the stools are either soft (containing soft curds) and oily in appearance or of a creamy consistence, or, especially in cases of long duration, gray or grayish yellow, hard and dry, forming the so-called "soap-stools." Sometimes the stools are watery, strongly acid, causing severe irritation of the buttocks. The vomitus is also strongly acid. The lips are often cherry-red in color.

In *sugar* indigestion vomiting is less common than in fat indigestion, but if it does occur, the vomitus, like the feces, is acid in reaction and often presents the characteristic odors of lactic, acetic or succinic acid. The stools are usually thin, often mixed with mucus, light or dark green, and very irritating to the buttocks. In severe cases there may be high fever, with other symptoms of acute intoxication.

Starch indigestion may give rise to loose, brown stools, mixed with

---

*The following fairly represents the *average capacity of the infantile stomach*: At the end of the first week, 1 ounce; the second week, 2 ounces; first month, 3 ounces; second month, 4 ounces; fourth month, 5 ounces; sixth month, 6 ounces; eighth month, 7 ounces; tenth month, 8 ounces; twelfth month, 9 ounces; fourteenth month, 10 ounces.

mucus, changing into blue color on addition of iodine. Infants fed exclusively on starch food slowly develop athrepsia.

An excess of *casein* usually gives rise to large, often tough, curds in the vomitus and stools, neutral or slightly acid in reaction and free from any characteristic odor. In some cases the stools are loose, mucous, brown in color, and musty in odor.

The management of the aforementioned digestive disturbances, in a way, is self-evident: we have to reduce temporarily the offending food element in the infant's diet, which must either be reduced in quantity or eliminated entirely. Skimmed milk and cereals should be given in fat indigestion: diluted skimmed milk or *Eiweissmilch* in carbohydrate indigestion; or condensed milk, well-diluted boiled milk, or weak mixtures of *Eiweissmilch* with the addition of malt dextrin in digestive disturbances due to an excess of protein. Of course, with disappearance of the symptoms the required fat, carbohydrate- and protein-proportions of the food are gradually to be resumed. (See also "Dyspepsia" and "Acidosis.")

Cow's Milk Substitutes

Malt Soup.—Two ounces of wheat-flour are slowly and thoroughly mixed with one pint of milk, and strained through gauze. In a second vessel 3 ounces of thick malt are dissolved in a quart of warm water to which had been added 15 grains of carbonate of potassium.* Now both solutions are mixed together and heated very slowly up to a boil. As the children improve the water may gradually be reduced to a pint. Malt soup is often particularly beneficial in underfed, dyspeptic and rachitic babies. If well tolerated it may be continued for several months.

It is advisable, however, gradually to replace the malt soup by ordinary milk mixtures, and other foods (p. 60).

Condensed Milk.—Where the principal difficulty consists in incapacity to digest cow's milk casein, condensed milk† will be found to act kindly, since the consistency of the coagulum of condensed-milk casein formed in the infantile stomach greatly resembles that of human milk. It has also the advantages of being inexpensive and not as readily subject to contamination as ordinary cow's milk. However, containing as it does about 51 per cent of sugar, and requiring eight to ten times dilution to approximate the sugar content of human milk,

---

*Malt soup extracts are now procurable in every reliable pharmacy.
†Approximate Composition of Canned Condensed Milk:

Protein	Sugar	Fat	Salts	Water
8.00	51.00	7	1.50	32.00

Fresh condensed milk contains only 10 per cent of sugar.

the simultaneous reduction (by dilution) of the fat and proteid contents to about 1 per cent and 1¼ per cent respectively, renders condensed milk too poor in quality to serve as an ideal infant food. Indeed, it is usually found that infants over three months, fed on diluted condensed milk, soon contract rachitis. Nevertheless, as a temporary food, especially during the summer months or on a long journey, it is invaluable. As already suggested, condensed milk should be administered in quantities appropriate for the infant's age, in dilution with from eight to ten or even twelve parts of plain or cereal water. The deficiency of fat may be supplemented by the addition of cream.

Whey.—Where the digestive capacity of casein is greatly at fault, we may temporarily resort to whey feeding. Whey is obtained by adding to a pint of fresh warm (100° F.) milk, two teaspoonfuls of essence of pepsin. After it stiffens, beat up the curd with a fork and strain through a few layers of gauze, so as to withhold the coagulated casein. The decanted liquid contains approximately:

	PROTEIN	SUGAR	FAT
Lactalbumin	0.9%	4.5%	0.5%
Casein	0.3%		

By adding a little cream to overcome its deficiency and employing a cereal diluent instead of plain water, the whey mixture is amply nutritious to sustain an infant's vitality for several weeks.

*Buttermilk.**—This is prepared by thoroughly mixing, in a suitable agate vessel, one quart of fresh, rich milk, with a pint or less of water, a pinch of salt, and the pure lactic acid culture (any one of the pure mercantile lactic bacilli tablets answers the purpose). The vessel is covered with a thin cloth and allowed to stand in the room (70° to 80° F.) for from eighteen to twenty-four hours. It is now placed on ice until needed. For infant feeding we add to every quart of buttermilk a flat tablespoonful of wheat-flour and two tablespoonfuls of cane sugar and allow the mixture to *boil* over a low fire, for two to three minutes, with constant stirring. The food is now poured, in quantities varying with the age of the patient, into sterilized bottles, properly corked, and placed on ice until used. The mixture is indicated especially in cases requiring a high percentage of protein and a low percentage of fat, *e. g.,* gastroenteritis and fat indigestion.

Eiweissmilch (Albumin-, Protein-, or Casein- Milk).—This food, originally recommended by H. Finkelstein and L. Meyer, is gradually being accepted by the profession as an ideal food in the management of fermentative dyspepsia and nutritional disturbances from intolerance of

---

*Composition: Water: 90.27; protein: 4.06; fat: 0.93; sugar: 3.73; salts: 0.67.

milk. It consists of 2.50 per cent of fat, 1.5 per cent of sugar, 3 per cent of protein, and 0.50 per cent of salts, and is prepared as follows: One liter of warm milk is treated with 15 grams of essence of pepsin, and allowed to stand in a water bath at 107.6° F., until a curd is formed. This mass is poured into a linen bag and allowed to filter for about half an hour, and while gradually adding half a liter of water the curd is pressed through a fine sieve two or three times by means of a wooden spoon. To this milk-like mixture we next add half a liter of butter-milk. Finkelstein and Meyer were prompted to suggest the *Eiweissmilch* after establishing the facts that nontoxic fermentative dyspepsia is due principally to abnormal fermentation of the carbohydrates (not the casein!) of the infant food, and that fat forms a disturbing element only when preceded by sugar fermentation. Albumin milk should be given in quantities of about 1½ to 2 ounces every three or four hours. In very young infants it may at first be diluted with an equal quantity of plain water and later barley water. As the patient improves, it is advisable to increase the amount of the *Eiweissmilch* and to strengthen it also by the addition of 1 per cent of maltose, or malt dextrin. After full recovery from the disease, *Eiweissmilch* feeding is gradually discontinued. Protein milk is now obtainable in powder form.

Dry Milk.—A number of clinicians have for some time been advocating the use of dry milk in infant feeding, especially in cases of difficult digestion and on long journeys. The approximate composition of whole dry milk is as follows: Fat 25 per cent, lactose 40 per cent, protein 28 per cent, salts 7 per cent and moisture 5 per cent. It is usually administered in 1 to 3 or 1 to 2 dilutions, with plain, boiled, or cereal water, in the same manner as fluid milk. Its prolonged use is contraindicated, even though some pediatrists claim that dry milk is not productive of scurvy or rachitis.

Mode of Manufacture.—The principal processes by which dried milk is made today are briefly as follows:

A. Milk is fed in a thin stream over two steam-heated cylinders or drums, about one-eighth of an inch apart and revolving in opposite directions. The milk exposed to the heat of the cylinders dries as a thin film and comes off the revolving cylinder as a sheet, which is easily crushed into a fine powder. The cylinders, which are some sixty inches long and 24 inches in diameter, are charged with steam under two or three atmospheres of pressure causing the heating surfaces to have a temperature of about 250 to 280° F. This process, known as the Just patent in the United States and as the Just-Hatmaker patent in England, is said to be the invention of J. R. Hatmaker, of London.

B. The milk is first pasteurized and then condensed in the vacuum pan at a low temperature (130° F.) to about one-fourth of its bulk. This condensed product is forced under high pressure through minute openings in a metal disk into a hot-air chamber. The atomized liquid surrounded by a current of hot air instantly dries and falls to the bottom of the chamber as a snowy powder, the moisture rising as a cloud of steam. The mixture of the liquid and air in the evaporating chamber is stated to be about 180° F. This method was originally developed in France and is called there and in England the Bevenot de Neveu process. In this country it is known as the Merrill-Gere process.

C. A third method of making dried milk, by reducing it to approximate dryness in a vacuum pan equipped with a mechanical stirrer, is also used in this country. It has the advantage of exposing the milk to a low though prolonged temperature.

Proprietary Milk Modifiers and Milk Foods.—We distinguish two kinds of proprietary foods—milk modifiers and so-called milk foods. Neither of them contain a sufficient amount of nutrient elements to supply the needs of the baby for life and growth for any length of time; they are useful, however, in digestive disturbances and "milk idiosyncrasy," and to bridge over an acute siege of sickness. The mercantile milk modifiers furnish soluble carbohydrates, free starch, or predigested proteids in small quantities, and thus save the trouble of home preparation of suitable diluents. Their prolonged exclusive use is frequently followed by scurvy and rickets.

Peptonized Milk.—The use of peptonized milk is nowadays limited chiefly to feeding of children of very low vitality, in whom the powers of digestion are in abeyance, *e. g.*, high fever, coma (administered in the form of nutrient enemata, or by gavage), pylorus stenosis, etc.

Mode of Preparation.—Mix in a quart bottle one pint of fresh milk with 4 ounces of cold water containing 5 grains of pancreatic extract and 15 grains of sodium bicarbonate, or the contents of one of Fairchild's peptonizing tubes. Place the bottle in a pot of hot water and maintain its temperature at about 115° F., either for about twenty minutes ("partial" peptonization) or two hours ("complete" peptonization). Shake the bottle from time to time. When the mixture is ready, give it, either pure or diluted, in quantities suitable for the age of the child. Keep it on ice until used.

Weaning the Baby and Its Feeding Thereafter

Ordinarily it is not advisable to nurse an infant beyond ten or eleven months old. As exceptions to this rule, we may mention the very hot summer months, acute diseases, difficult teething, etc., when a complete change in feeding is prone to prove hazardous to the

child's health. It is preferable to wean a baby gradually, by substituting bottle- for breast-feedings, and to continue partially to nurse it, until the infant has learned to submit to the inevitable, and thrives well on the new food.

Feeding of Infants Over Seven Months Old.—When the normal infant reaches the age of seven months or thereabouts, nature announces the urgency of a change in the dietary—from liquid to solid—by hastening the eruption of the lower and upper incisors. At this age, also, salivary digestion is fully established, so that an allowance, once or twice a day, of a crust of stale or toasted bread, or zwieback, certainly can do no harm. As at this period of life the tendency to rickets is very pronounced, the dietary should be gradually improved upon by the addition of small quantities of cereals, a teaspoonful or more of fresh, soft-boiled egg, strained chicken, mutton, or beef soup, with fresh vegetables (*e. g.*, carrots, potatoes, etc.), orange or pineapple juice, baked potato with some sweet cream or butter; and later (at about a year,) bread and butter, milk custards, cocoa, and occasionally finely scraped beef or chicken.

Of course the transition from an exclusive milk diet to a more or less mixed diet must be very slow and gradual. The effect of the change should be watched from day to day and week to week, always bearing in mind that milk is the ideal food for the infant and indispensable to the child up to the period of second dentition.

This fact should be strongly impressed upon those in charge of the child, as only too often, with the allowance of a semisolid diet, milk is crowded out entirely by an oversupply of thin soups, indigestible, proprietary "breakfast foods," and all sorts of sweets and fruit of poor quality, which sooner or later upset the child's digestive powers and arrest its growth and development—doing just the opposite of what the change of diet was intended for.

With the change in the diet it is also frequently observed that the infants refuse to drink milk. Inquiry into the cause usually reveals the fact that upon the advice of some artistically inclined neighbor— who thinks that the bottle effaces the child's "beauty lines"—and more generally upon the recommendation of the family physician, the child is forced to part with its bottle and nipple—its dear and faithful companions for the many months past. Why milk bottles are to be looked upon as an abomination for children *over* a year or so and as a salvation for those *under* this age, is to me a mystery. The mere facts that if given in a bottle, large quantities of milk are enjoyed by children up to four or five years of age; that if taken through a nipple, milk enters the stomach slowly, and, hence, is more easily digested, and finally, that during sickness milk (as well as water) is best administered

through a bottle, are ample justifications for the encouragement rather than the prohibition of the use of the bottle—provided, of course, that the bottles, as well as the nipples, are kept scrupulously clean; are sterilized, if you please.

The additional articles of food should be given at definite intervals, preferably together with the milk feeding. Thus, for example, with the ten o'clock bottle the child should receive the soft-boiled or poached egg and crackers; at two o'clock the meat broth and potato; at six o'clock, some cereal and bread and butter. Orange or pineapple juice may also be given between meals. The child should be taught to appreciate that to get other foodstuffs it must drink its allowance of milk.

DIET FOR CHILD
FROM 18TH TO 24TH MONTH
Breakfast

1. Juice of 1 sweet orange
   or
   Pulp of 6 stewed prunes
   or
   Pineapple juice (fresh or bottled) 1 ounce.
2. *Dessert*: apple sauce, prune pulp, with stale lady-fingers or graham wafers.
   top milk (top 16 ounces) sweetened or salted. A glass of milk, bread and butter.
   Note: If constipated give the fruits half-hour before breakfast with water; if not, they may be given during the forenoon.
   Raw fruit juice must be given either half-hour before or half-hour after milk.

Forenoon

A glass of milk with two toasted biscuits or zwieback or graham crackers.

Dinner

1. Broth or soup made of beef, mutton, or chicken, and thickened with peas, farina, sago or rice
   or
   Beef juice with stale bread crumbs; or clear vegetable soup with yolk of egg
   or
   Egg soft boiled, with bread crumbs, or the egg poached, with a glass of milk.
2. *Dessert*: apple sauce, prune pulp, with stale lady-fingers or graham wafers
   or
   Plain puddings: rice, bread, tapioca, blanc-mange, junket or baked custard, bread and butter.

3 p. m.

A cup of milk with biscuits.

Supper

An egg, glass of milk, zwieback and custard, or stewed fruit.
Total milk in 24 hours, 1 quart.

DIET FOR CHILD
FROM TWO TO THREE YEARS
Breakfast

1. Juice of 1 sweet orange
   or
   Pulp of 6 stewed prunes
   or
   1 ounce pineapple juice (fresh or bottled)
   or
   Apple sauce (warm).

2. A cereal such as oatmeal, farina, cream of wheat, hominy or rice, slightly sweet-
   ened, or salted, as preferred, with the addition of top milk.
     or
   A soft boiled or poached egg with stale bread or toast.
3. A glass of milk.
   Note: If constipated give the fruits ½ hour before breakfast with water; if not,
   they may be given during the forenoon.
   Milk and raw fruit juice must not be given at same meal.

Dinner

1. Broth or soup made of chicken, mutton or beef, thickened with arrowroot, split
   peas, rice, or with addition of the yolk of an egg or toast squares.
2. Scraped beef or white meat of chicken or broiled fish (small amount) bread and
   butter
     or
   Mashed or baked potatoes with fresh peas or spinach or carrots.
3. *Dessert*: apple sauce, baked apple, rice pudding, junket or custard.

3 p. m.

A cup of milk with biscuits.

Supper

1. A cereal or egg with stale bread or toast and butter. Cup of cocoa.
     or
   Bread and butter and milk, or bread and butter and cocoa, or bread and custard.
2. Stewed fruit.

Feeding of Children from Four to Six Years Old.—The dietary of
children over four years old is practically identical with that just men-
tioned, except that the quantity of the food should be more liberal, the
fruit may be given raw, and that the between-meals milk allowance
should be dispensed with. Occasionally the child may receive home-
made cake, a little ice cream and other condiments of good quality. All
these foodstuffs, however, should be given with regular meals.

Average Composition of Common American Food Products*

Food materials (as purchased).	Refuse	Water	Protein	Fat	Carbo-hydrates	Ash	Fuel value per pound
	p. c.	p. c.	p. c.	p. c.	p. c.	p. c.	Calories.
Sirloin steak...........	12.8	54.0	16.5	16.1	....	.9	975
Round steak...........	7.2	60.7	19.0	12.8	....	1.0	890
Veal, leg cutlets.......	3.4	68.3	20.1	7.5	....	1.0	695
Veal, breast...........	21.3	52.0	15.4	11.0	....	.8	745
Mutton, leg, hind......	18.4	51.2	15.1	14.7	....	.8	890
Mutton, loin chops.....	16.0	42.0	13.5	28.3	....	.7	1,415
Lamb, breast..........	19.1	45.5	15.4	19.1	....	.8	1,075
Pork, loin chops.......	19.7	41.8	13.4	24.2	....	.8	1,245
Ham, smoked..........	13.6	34.8	14.2	33.4	....	4.2	1,635
Bacon, smoked.........	7.7	17.4	9.1	62.2	....	4.1	2,715
Soup, beef.............	....	92.9	4.4	.4	1.1	1.2	120
Soup, tomato..........	....	90.0	1.8	1.1	5.6	1.5	185

*After Nelson's Perpetual Loose Leaf Encyclopædia.

AVERAGE COMPOSITION OF COMMON AMERICAN FOOD PRODUCTS—CONT'D

Food materials (as purchased).	Refuse	Water	Protein	Fat	Carbo-hydrates	Ash	Fuel value per pound
	p. c.	p. c.	p. c.	p. c.	p. c.	p. c.	Calories.
Chicken, broilers.......	41.6	43.7	12.8	1.4	......	.7	305
Fowls	25.9	47.1	13.7	12.3	......	.7	765
Cod, dressed...........	29.9	58.5	11.1	.2	......	.8	220
Mackerel, whole.........	44.7	40.4	10.2	4.2	......	.7	370
Salmon, canned.........	......	63.5	21.8	12.1	......	2.6	915
Oysters, "solids".......	......	88.3	6.0	1.3	3.3	1.1	225
Lobsters	61.7	30.7	5.9	.7	.2	.8	145
Hens' eggs.............	a11.2	65.5	13.1	9.3	......	0.9	635
Butter	......	11.0	1.0	85.0	......	3.0	3,410
Whole milk............	......	87.0	3.3	4.0	5.0	.7	310
Buttermilk	......	91.0	3.0	.5	4.8	.7	160
Cream	......	74.0	2.5	18.6	4.5	.5	865
Cheese, full cream.......	......	34.2	25.9	33.7	2.4	3.8	1,885
Entire-wheat flour.......	......	11.4	13.8	1.9	71.9	1.0	1,650
Flour, white...........	......	12.0	11.4	1.0	75.1	.5	1,635
Corn meal.............	......	12.5	9.2	1.9	75.4	1.0	1,635
Oat breakfast food.....	......	7.7	16.7	7.3	66.2	2.1	1,800
Rice	......	12.3	8.0	.3	79.0	.4	1,620
Tapioca	......	11.4	.4	.1	88.0	.1	1,650
Bread, white...........	......	35.3	9.2	1.3	53.1	1.1	1,200
Bread, Graham.........	......	35.7	8.9	1.8	52.1	1.5	1,195
Cake	......	19.9	6.3	9.0	63.3	1.5	1,630
Soda crackers	......	5.9	9.8	9.1	73.1	2.1	1,875
Molasses	......	......	......	......	70.0	......	1,225
Candy, plain...........	......	......	......	......	96.0	......	1,680
Sugar, granulated......	......	......	......	......	100.0	......	1,750
Beans, dried...........	......	12.6	22.5	1.8	59.6	3.5	1,520
Cabbage	15.0	77.7	1.4	.2	4.8	.9	115
Celery	20.0	75.6	.9	.1	2.6	.8	65
Onions	10.0	78.9	1.4	.3	8.9	.5	190
Peas, shelled	......	74.6	7.0	.5	16.9	1.0	440
Potatoes, white.........	20.0	62.6	1.8	.1	14.7	.8	295
Potatoes, sweet........	20.0	55.2	1.4	.6	21.9	.9	440
Tomatoes	......	94.3	.9	.4	3.9	.5	100
Apples, fresh..........	25.0	63.3	.3	.3	10.8	.3	190
Apples, dried..........	......	28.1	1.6	2.2	66.1	2.0	1,185
Bananas	35.0	48.9	.8	.4	14.3	.6	260
Grapes	25.0	58.0	1.0	1.2	14.4	.4	295
Muskmelons	50.0	44.8	.3	......	4.6	.3	80
Oranges	27.0	63.4	.6	.1	8.5	.4	150
Strawberries	5.0	85.9	.9	.6	7.0	.6	150
Watermelons	59.4	37.5	.2	.1	2.7	.1	50
Raisins	10.0	13.1	2.3	3.0	68.5	3.1	1,265
Chestnuts	24.0	4.5	8.1	5.3	56.4	1.7	1,385
Peanuts	24.5	6.9	19.5	29.1	18.5	1.5	1,775
Walnuts, English.......	58.1	1.0	6.9	26.6	6.8	.6	1,250
Chocolate	......	5.9	12.9	48.7	30.3	2.2	2,625
Cocoa, powdered	......	4.6	21.6	28.9	37.7	7.2	2,160
Cereal coffee, infusion (1 part boiled in 20 parts water)........	......	98.2	.2	......	1.4	.2	30

II. HYGIENE AND SANITATION

Next to suitable nutrition, hygiene and sanitation play the most important rôle in the preservation of good health. It is within the province of the physician's duties to formulate, to those intrusted with the care of the child, rules and regulations as to its cleanliness and comfort, mode of clothing, time for sleeping, airing, bathing, rest and exercise, both during health and disease. Without the advice and supervision of the physician, the nurse or mother is only too apt either to overdo or underdo, *i. e.*, in both events do irreparable damage to the health and welfare of the child. Unfortunately blind credulity, stupid mysticism and absurd fatalism still reign supreme, the great strides in science and adventure notwithstanding.

General Care of the Newborn and Older Children

The Newborn.—Immediately after birth the infant instinctively, by its shrill cry, announces its demand for protection against the sharp change of atmosphere and surroundings. Therefore, after dressing its navel (p. 221), washing its eyes and mouth with a saturated boric acid solution, and instillation into each eye of one drop of a 2 per cent solution of nitrate of silver, the baby should be wrapped in a warm woolen blanket and placed in a warm, darkened, but airy, quiet room, and left to rest for a few hours. It should then be sponged off with warm soap water, dressed, given a little clean water, and, the condition of the mother permitting, put to the breast (p. 44). Whenever possible, the child's crib should be kept in a room apart from that of the mother, so that the latter is not disturbed by the possible uneasiness experienced by the baby. As lactation is usually not fully established before the third or fourth day after labor, the infant should, in the meantime, several times daily receive a few teaspoonfuls of plain or slightly sweetened warm water or of a mild carminative, such as fennel-seed tea, to satisfy its thirst and hunger.

Sleep.—The normal newly born baby sleeps practically all the time except the brief periods occupied with nursing, diapering, and dressing. If the baby is well developed and strong, it should be left to sleep until it wakes up of its own accord from hunger; if delicate, it should be aroused every two or three hours during the day, and once at night, made to cry a little to help to expand its lungs and put to the breast for from ten to twenty minutes. At six weeks the infant needs twenty hours of sleep; at three months, eighteen; at one year, sixteen, and from two to four years, fourteen hours of sleep. All children should get accustomed to sleep uninterruptedly (except for one nurs-

ing in the middle of the night in early infancy), from seven in the evening until seven o'clock in the morning, and one hour each some time between seven and twelve o'clock in the forenoon and two and seven in the afternoon.

Sleeplessness in the infant is ordinarily due to intestinal colic or other pain, discomfort from soiled diapers or faulty dressing (over-heating by superabundance of clothes, etc.), noise in the room, lack of ventilation, bad habits, such as rocking, or keeping an empty nipple in the mouth, etc. Repeated waking is frequently due to over- or under-feeding.

Bathing.—In view of possible local or systemic infection (p. 219) through the umbilical rest, and the advisability of keeping the latter perfectly dry, the full tub bath should be withheld until the navel has completely healed. The same applies for circumcision wounds. In the meantime the infant should receive at least one sponge bath a day, to be given as gently as possible, since the infantile skin is very delicate, very apt to be abraded on rough handling, and readily becomes subject to divers skin affections.

In the absence of the aforementioned or other contraindications, every child, in addition to local cleansing as frequently as necessity arises, should receive a tub bath once a day, preferably at bedtime. The water used should be free from visible impurities, and obtained from sources inaccessible to pollution. The temperature of the water should range between 95° F. and 98° F., the latter for infants under six months, and cooler water for older ones. Fat babies tolerate much lower temperatures, but I see no special benefit to be derived from the use of bath water under 95° F. unless it be in the open sea or ocean (which is permissible in children over three years of age), where the saline ingredients and forceful current exert a stimulating, refreshing effect upon the system and thus counteract the depression produced by the sudden lowering of the body temperature. If cool bathing is desirable, it is better to place the child in warm water and either gradually cool off the water while the child is in the tub or use a cold shower. The bath should be followed by thorough drying of the body and gentle friction. Care should be exercised in the selection of pure, nonirritating bathing soap, lest its irritating ingredients may prove a source of annoying skin eruptions. For the same reason and, furthermore, owing to the fact that they are apt to harbor dirt and disease, the use of sponges is to be deprecated.

Clothing.—Infants should be clothed warmly and simply, free from fancy frocks and frills, strings and bows, that embarrass free motion,

breathing, sleeping and eating. The underwear should be made of silk or thin flannel. The abdomen should be protected against being chilled by a flannel band. The consistency of the outer clothing should vary with the changes of the weather and season of the year. The feet of infants should at all times be kept warm, if necessary, by means of a hot-water bag. The night clothes should be loose and warm, and consist, in addition to a small silk or flannel shirt, Canton flannel or stockinet diaper and the belly-band, of a nightshirt in the form of a "bag" that buttons around the neck and can be closed at the feet by means of drawstrings. In this manner the unnecessary piling up of blankets, to keep the baby from uncovering, can be advantageously dispensed with.

Older children should gradually get accustomed to light clothes— linen or silk undergarment, linen or woolen suit or dress, and for the winter a warm top coat and cap—but no collars or neck mufflers. A woolen union suit with feet for the night. Special attention should be paid to the selection of shoes. They should comfortably fit the feet and allow spreading of the toes. The stockings should be fastened to the drawers, as garters are apt to interfere with the blood circulation of the lower extremities. The corset should be prohibited in girls under fourteen.

Airing.—Fresh, pure air is the panacea for good health, the cure of all bodily ills. Thus far it is nonassessable, nontaxable, and hence should be inhaled *ad libitum*—while this freedom lasts. Weather permitting, it should be inhaled out of doors, otherwise indoors—in properly ventilated rooms. The newborn should be taken outdoors in the summer when it is two weeks old, in the spring and fall at one month and in the winter at two months of age or later. It should be suitably dressed and protected from undue exposure to the sun and wind and severe cold. It is foolhardy to expose an infant to marked atmospheric changes without proper shelter, merely for the purpose of "hardening" it. Its first airing should last from fifteen to thirty minutes, and as it grows older the airing time should be lengthened, so that, weather permitting, the child may live outdoors the greater part of the day from sunrise until sunset. Slight rain or snow forms no hindrance to taking the baby outdoors, although in such weather delicate babies do better if aired indoors, in front of open windows and dressed as for outdoors.

Exercise.—A healthy infant, if not immobilized by burdensome clothes, begins to take physical exercise soon after birth. It kicks, moves its arms and head and exercises its thoracic muscles while crying lustily, especially when feeding time approaches. It should be

picked up in the arms at every nursing to insure change of position. At about four months of age the baby is able to hold its head erect; it may then be gradually trained to sit upright upon the arm of the nurse, its back and head well supported. As it reaches the age of seven or eight months, the infant may be seated alone in a baby-chair supported with pillows at the back and sides. When it shows an effort to creep, it may be placed upon the floor, which should be well covered by thick carpet or a blanket, preferably within a small portable "creeping pen," and allowed to roam about for half an hour at a time once or twice a day. Less freedom should be granted an infant in its first attempts to stand or walk. These practices should not be encouraged in babies under one year of age, nor in older children who show a tendency to bony curvatures and rickets. In the beginning they should not be allowed to stand or walk, especially if unsupported, for more than a few minutes at a time. But, as they grow older and stronger, they are gradually permitted to enjoy shorter or longer outdoor walks and to romp merrily, giving vent to that characteristic boundless joyousness of early childhood which is blessedly ignorant of the pangs and pains of later life.

Older children, like infants, should spend the greater portion of the day outdoors in parks and playgrounds and engage in amusing games and light calisthenics which will keep them from harm and mischief. It is opportune on this occasion to emphasize the danger of overindulgence in the practice of gymnastics, especially in children of school age—a period of life which is coincident with prevalence of communicable diseases and their grave sequelæ, particularly cardiac involvement.

It is the duty of the physician to impress upon those under his care that while moderate exercise—especially walking, skating and horse-back riding; the daily use, for about fifteen minutes at a time, of light wooden dumb-bells, light clubs or wands; the practice of breathing (p. 439), of swinging of the body from a swinging bar or rings and straps,—will do much for the development of delicate and narrow chests and to prevent and straighten curvatures of the spine, stooping of the shoulders, and the like (and should be encouraged), violent sports, like racing, rough baseball- and football-playing, leaping, prolonged swimming and similar severe exercises indulged in to excess, will sooner or later lead to cardiac hypertrophy with its consequences.

Nursery.—As infants and older children spend about two-thirds or more of their time of life in the nursery, provisions must be made that the room is spacious and airy, dry and sunny, that its air is fresh and pure, free from obnoxious odors, gases, dust and smoke. To thrive

well an infant requires about 1000 cubic feet of air space. The room should not be crowded with dust gatherers, *i. e.*, overabundance of furniture, toys, heavy hangings, carpets, rugs, pictures, etc. The temperature of the room should be about 70° F. during the day and about 65° F. during the night. Whenever possible, it should be heated from an open fireplace or hot-air furnace. Steam heat or gas often vitiates the air. To insure proper ventilation, it is advisable to keep the windows more or less open from top and bottom most of the time unless the outdoor temperature is below 35° F. The windows and doors should be widely opened while the child is outdoors, otherwise ventilation should be accomplished with the doors closed to avoid draughts. For the latter purpose one of the many ventilating devices on the market will prove very serviceable.

Financial circumstances permitting, every child should have a separate room, if possible, situated one floor above the ground. Of course, this is rarely attainable in the dingy apartments of overcrowded cities. Physicians should insist, however, on every child having a separate bed in order to minimize the danger of transmitting communicable diseases from the sick to the healthy child.

The Sick-Room.—The hygienic suggestions just made in reference to the nursery apply with greater force to the sick-room. If possible, the latter should be situated on a different floor from the living apartments. From a sanitary as well as economic point of view it is essential to have the sick-room cleared of curtains, tapestries, superfluous furniture, carpets, etc., so as to facilitate keeping the room perfectly clean, and to prevent pathogenic germs becoming firmly imbedded in those articles. The floor and furniture of the sick-room should be wiped off with a damp cloth instead of dusted or swept.

An anteroom is a useful addition to a sick-room especially when the patient is suffering from a communicable affection, as it enables the nurse to disinfect the dishes, soiled bedclothes, linen, etc., and to prepare some of the patient's food.

When the isolation-period of the patient is over, the sickroom, anteroom and their contents must undergo very thorough cleaning and disinfection.

Quarantine and Disinfection.—In order to prevent spreading of communicable diseases from one individual to another, we have to resort principally to the following prophylactic measures:

1. Isolation of the patient.
2. Disinfection of the patient's excretions, fomites, etc., coming in contact with the pathogenic microorganisms.

3. Exclusion of visitors and domestic animals, such as cats and dogs, and destruction of other germ carriers, e. g., mosquitoes, flies and fleas.

1. **Isolation of the Patient.**—This is the most essential and efficient mode of prevention of transmission of disease. The isolation to be effective must begin early and be complete. In hospitals and asylums every child should be isolated in an observation ward for at least three days before being permitted to mingle with the other inmates; in private families isolation should be enforced with the earliest appearance of tangible symptoms of the specific affection. As those coming in close contact with the patient are apt to carry the disease from the sick to the well, it is imperative to isolate the nurse together with the patient and to forbid any member of the family to stay around the sick-room or make herself generally useful, unless on entering the sick-room she dons a clean gown and cap, and before leaving it washes her hands and forearms with soap and water and removes the gown and cap. These latter rules should apply also to the physician.

In a private dwelling, and especially in houses where a room is reserved for the sick, perfect isolation can readily be insured. In crowded tenement rooms, however, with people in poor circumstances, all attempts at isolation almost invariably fail, and where the spreading of a grave, epidemic affection is concerned (e. g., smallpox, cerebrospinal meningitis), should not at all be attempted. In such cases it is best to remove the patient to a hospital for contagious diseases.

The period of isolation varies, of course, with different diseases and the degree of severity. The following suggestions will meet the ordinary requirements as to the period of isolation and the principal mode of prophylaxis:

In typhoid fever, while the disease lasts. (Disinfection of excreta; protection against flies, fleas, lice, etc.)

In typhus fever, while the disease lasts. (Same as in typhoid.)

In miliary tuberculosis, while the disease lasts. (Disinfection of excreta.)

In epidemic cerebrospinal meningitis and poliomyelitis, while the disease lasts. (Disinfection of discharges.)

In yellow fever, while the disease lasts. (Destruction of mosquitoes.)

In relapsing fever, while the disease lasts. (Destruction of insects.)

In influenza, pneumonia and pulmonary tuberculosis, while the diseases last. (Disinfection of discharges.)

In bubonic plague, about one week after termination of the disease. (Destruction of vermin, especially rats; disinfection of excreta.)

In cholera Asiatica and epidemic dysentery, one week after termination of the disease. (Disinfection of excreta; avoidance of pollution of water, milk, etc.)

In smallpox, six weeks. (Vaccination; disinfection of discharges.)

In chickenpox, one week. (Disinfection of discharges and skin.)

In measles, two weeks. (Disinfection of discharges and skin.)

In German measles, two weeks. (Disinfection of discharges and skin.)

In diphtheria, as long as diphtheria bacilli abound in the throat. (Disinfection of discharges.)

In scarlet fever, while the desquamation lasts. (Disinfection of discharges and skin.)

In whooping-cough, while whoop or vomiting lasts. (Disinfection of expectoration.)

In mumps, two weeks. (Disinfection of sputum.)

In erysipelas, two weeks. (Disinfection of the skin; antiseptic dressing.)

In gonorrheal ophthalmia or urethritis, while gonococci are found in the discharges.

Before leaving the isolation room, the patient should receive a cleansing, hot soap-water bath (including thorough scrubbing of the scalp, ears, finger- and toe-nails), and dressed anew with freshly disinfected clothing.

2. **Disinfection of Excreta, or Fomites, etc.**—In order to be on the safe side, the nurse should be instructed to disinfect the stools, urine, vomitus, sputum, and nasal, aural, conjunctival and vaginal discharges of the patient, regardless of whether or not they carry contagious matter.

For Excreta.—Chloride of lime in powder or in solution. Four ounces of lime to one gallon of soft water. A sufficient quantity of this solution should be thoroughly mixed with the feces, urine, sputum, etc., and allowed to stand for about three hours before emptying.

Sputum is best collected in paper cups, or small cloths and immediately destroyed by fire.

Bichloride of mercury in solution 1:500—a 7½ grain tablet in a pint of water. Copper sulphate in solution (5 per cent). Zinc sulphate in solution (10 per cent). Cresol or creolin in solution (5 per cent).

For Clothing, Bedding, Linen, etc.—Destruction by fire—the safest measure. Exposure to dry heat at a temperature of about 300° F., or moist heat at 212° F., for two hours. Boiling for at least half an hour. Immersion in a bichloride solution (1:2000) for about three hours. Fumigation by formaldehyde. (See below.)

For the Hands, General Body, Dishes, etc.—Labarraque's solution (chlorinated soda, 10 per cent). Bichloride of mercury in solution (1:1000). Permanganate of potash in solution (1 ounce to a quart of water). Formaldehyde in solution (1:200).

For Rooms, Furniture, Mattresses, etc.—Fumigation by Formaldehyde Gas.—It may be employed in concentrated powdered form or in pastels. For small rooms the ordinary Shering lamp, which is constructed for vaporizing formaldehyde pastels will suffice. For large hospital wards,

however, the *"formaldehyde-potassium-permanganate method"* is best. It is of advantage to use a container consisting of a large open vessel protected from losing its heat by some nonconducting material such as asbestos. But one can get along almost equally as well by using a large milkpail set in a wooden bucket.

The infected room should be made as air-tight as possible by snugly closing the windows and doors (keyholes, ventilators, fireplaces, etc.) by means of cotton or cloths. All articles intended for disinfection are freely exposed (mattresses, pillows, boxes and drawers should be opened).

The fumigating apparatus is placed in the center of the room: 6¾ ounces of potassium permanganate (for each 1000 cubic feet of room space) are put in the container; and 16 ounces of a 40 per cent formaldehyde solution (for each 1000 cubic feet of room space) are poured on top of the permanganate. The operator now quickly leaves the room, and closes the door or window. The room should remain tightly closed for about ten hours.

After disinfection the disagreeable odor of the formaldehyde may be removed by sprinkling the room with ammonia water, and thorough ventilation.

Fumigation with Sulphur.—The procedures are the same as with formaldehyde. The sulphur, about 3 pounds for a room 10 feet square, is placed in an iron pan, supported by bricks and set in a tin vessel with water. The sulphur is ignited by live coals or a tablespoonful of alcohol lighted by a match. Sulphur fumigation should not wholly be depended upon after grave epidemic affections.

Finally, it is well to bear in mind that sunlight is a disinfectant of great efficiency, and that prolonged exposure to its rays will materially aid in rendering rooms and fomites free from infectious matter.

III. IMMUNIZATION—ACQUIRED IMMUNITY. BIOLOGIC DIAGNOSIS AND THERAPEUTICS

Medicine is rapidly reaching the goal of its highest ambition, the prevention and control of communicable diseases by "Nature's method," *i. e.*, immunization. Stupid skepticism and boundless enthusiasm are gradually yielding to deliberate experimentation and experience, and it does not require a very great stretch of imagination to predict that in the near future every communicable affection will be successfully resisted and combated by an antagonist evolved by the causal microorganism.

In order to obviate unnecessary repetition we shall briefly describe the biologic products at present in use for diagnostic, protective and therapeutic purposes, and the results thus far achieved.

Variola Vaccine

With the enforcement of vaccination by all civilized nations, smallpox, the most loathsome pestilence, has practically been eradicated from every well-regulated community. The principle of vaccination is the introduction into the human body of a weakened and harmless form of vaccinia, cowpox, which renders the system immune (*i. e.*, creates enough of antibodies to resist the disease) to variola. The vaccine is obtained from the vesicles that form on healthy young heifers as a result of inoculation with the virus of cowpox.

Vaccination

In the absence of contraindications (p. 74) every child of from six to twelve months old should be vaccinated, and revaccinated about seven years later. It is preferable to vaccinate at a time when neither excessive heat nor cold prevails, *i. e.*, in May or October. The left arm at the insertion of the deltoid is usually chosen for the vaccination. In girls the leg may be preferred to avoid the possibility of an exposed disfiguring scar. The parts to be inoculated should be freely bared and cleansed with soap and water and thoroughly dried. When one inoculation is to be made, the epidermis should be abraded for about $\frac{1}{8}$ inch in diameter (until a serous exudate or a trace of blood occurs) by means of a sterile needle; when several inoculations are to be made, they should be fully $1\frac{1}{2}$ inches apart. About a drop of vaccine is then gently rubbed into the denuded surface and allowed to dry. In successful vaccination the inoculated area begins to redden and swell on the third or fourth day; on the fifth day a vesicle appears which gradually changes into an umbilicated pustule surrounded by a red areola. The pustule persists up to the eleventh or thirteenth day and then becomes covered by a scab. The latter remains stationary for about ten days longer, then falls off, leaving behind a red scar which gradually becomes white and glistening in appearance. The scar usually remains visible throughout life. Vaccination is associated with more or less marked constitutional symptoms. With appearance of the vesicle there is a slight rise of temperature; the child is restless, sleeps badly, loses its appetite, and shows other signs of indisposition. Some children react more strongly than others, but if the vaccine is pure, the vaccinator clean and careful and the inoculated area kept free from irritation and infection, all the constitutional symptoms disappear by the twelfth day. Under adverse circumstances (*e. g.*, old, impure lymph, defective asepsis, constitutional diseases) vaccination may be accompanied by very grave symptoms. The pustules may become very large, the redness in the vicinity very marked and extensive; the axillary glands very much swollen and pain-

ful; the whole arm very strongly infiltrated; the fever very high, up to 104° F.; and convulsions and respiratory and gastrointestinal symptoms may develop. Suppuration of the glands, phlegmonous processes, and even erysipelas may set in. Finally, vaccination may be accompanied by transient or genuine nephritis, and cases of scrofula, tuberculosis and syphilis are on record—undoubtedly preexistent, latent, but awakened by the acute inflammatory process. Occasionally the inoculation wound fails to cicatrize, continues to suppurate, or ulcerates. Children with a tendency to skin diseases may develop divers skin eruptions, such as erythema, eczema, lichen, impetigo, psoriasis, a purpura-like eruption (*purpura vaccinatoria*), general furunculosis, or, by transference (autoinoculation) of the vaccine virus to some diseased parts of the skin, produce general vaccinia. The latter may also develop—usually about the seventh or eight day—spontaneously, from within, independently of any external influences. The lesions, which may be discrete or confluent (grave), bear a certain resemblance to the regular vaccinal pox. In the same manner the vaccine may be carried to the eyes (*vaccine ophthalmia*), and cause serious trouble. In fact, inoculation pustules have been observed on different portions of the body, and even on the tongue. Furthermore, vaccinia may also be transmitted to other persons by means of infected articles in use, fingers, bed sheets, bath water, sponges, etc. Hence the importance of a protective dressing over the vaccination mark (clean sterilized linen, sewed to the sleeve, changed every day) from the time the vaccine has dried up to the falling off of the scab, and of keeping the child's nails very short and its hands very clean. Bathing should be interrupted from the fifth to fifteenth day. Moist boric acid dressings are useful to reduce the severe, local inflammatory process, and where the latter is grave, and the itching intense, a continuous, moist dressing with nitrate of silver ($\frac{1}{4}$ per cent) will prove especially beneficial. In delayed healing the wound should be cauterized with a 5 per cent to 10 per cent solution of nitrate of silver, and dressed like any other wound. Other complications arising should be treated according to indications.

Revaccination.—As already suggested revaccination should be performed about seven years after the first vaccination, a period of time after which the immunity against smallpox usually ceases. In case of epidemics revaccination should be resorted to more frequently. Revaccination is also indicated to modify an attack of smallpox. In successful revaccination the local and systemic manifestations are essentially the same as after the first vaccination except that they are much milder in form.

Contraindications to Vaccination.—It is not advisable to vaccinate infants under three months, and children of all ages who are suffering from severe acute and recurrent skin affections, local or general syphilitic or tuberculous (scrofular) lesions, and great debility.

Schick's Reaction* for Detection of Susceptibility to Diphtheria.—The outfit of the New York Health Department for the Schick reaction consists of a capillary tube (containing two minimum lethal doses for the guinea-pig of undiluted diphtheria toxin) and a small rubber bulb, and a bottle filled with 10 c.c. sterile physiologic salt solution (with 0.025 per cent carbolin acid). A. Zingher gives the following directions for its use: Break off one end of the capillary tube and push it carefully through the neck of the rubber bulb until it punctures the diaphragm within and enters the cavity of the bulb; then break off the other end of the tube. Hold the bulb between thumb and middle finger; place the index finger on the opening of the outer end of the bulb and expel the toxin in the saline solution. Rinse out the capillary tube by repeatedly drawing up saline and expelling it into the bottle, then cork the bottle and shake the diluted toxin. Inject exactly 0.15 c.c. of the solution (representing 1/50 minimum lethal dose for the guinea pig) *intracutaneously* on the flexor surface of the forearm or arm. The injection is made with an all-glass syringe and fine needle. Instead of a syringe Koplik and Unger have devised a hypodermic shaped needle (with a handle) which is dipped into the undiluted toxin and introduced intradermically. In the absence of antitoxin in the child's blood, or in the presence of only a very minute amount, insufficient for protection against diphtheria, a circumscribed area of redness and infiltration, from ½ to 2 cm. in diameter appears on the skin in from twenty-four to forty-eight hours, and persists for about a week leaving behind a brownish pigmentation. The positive reaction should not be mistaken for a pseudoreaction (which is due to proteins) that occasionally appears after the test. The pseudoreaction is earlier in its appearance as well as disappearance and is more infiltrated than the genuine reaction. About 40 to 50 per cent of the children react positively to the toxin test.† All those who do so and are ex-

---

*Discovered by Dr. Schick of Vienna in 1912.

†*Susceptibility of Various Ages to Diphtheria*
(as indicated by the Schick diphtheria-toxin skin test.)

Age	*Susceptible*
Under 3 months 15	per cent
3 to 6 months 30	per cent
6 months to 1 year 60	per cent
1 to 2 years 60	per cent
2 to 3 years 60	per cent
3 to 5 years 40	per cent
5 to 10 years 30	per cent
10 to 20 years 20	per cent
Over 20 years 12	per cent

posed to diphtheria infection, should immediately receive a prophylac-
tic dose of diphtheria antitoxin. The test is also very valuable in scar-
latina to determine the child's susceptibility to diphtheria, which forms
so frequent a complication during the course of the disease.

Antidiphtheritic Serum

Diphtheria antitoxin is the purified blood-serum of a horse that has
been rendered immune to diphtheria by a long course of treatment
with diphtheria toxin. It is specific in its effects, having lowered the
high (40 to 60 per cent) mortality from diphtheria to about 5 per cent
—if administered early and in ample quantity. Furthermore, those
exposed to diphtheria almost invariably escape infection by timely
administration of the serum. It is practically harmless if free from
admixture of virulent bacteria, and with introduction of the concen-
trated, high-grade preparations and the application of greater care
in handling and administration, the numerous disagreeable accompani-
ments (fever, multifarious eruptions, articular swellings, etc.) have
ceased to be as common and as severe as in former years.

The dose of antitoxin for ordinary cases of diphtheria should be
2,000 units for every year of the child's age up to five years, and 10,000
units as the average dose for older children. If urgent, the injections
may be repeated once or twice at intervals of from six to twelve hours.
Malignant, especially laryngeal, cases require double doses. For pro-
tective purposes a third of the ordinary dose usually suffices. The pro-
tection usually lasts from four to six weeks.

The antitoxin is administered by a sterile hypodermic syringe (or
the mercantile serum-containing syringes) by deep injection into the
anterior surface of the abdomen or thorax or outer surface of the
thigh, which are rendered aseptic by soap, water, ether and alcohol,
or tincture of iodine. The point of injection is subsequently sealed
by sterile adhesive plaster.

Diphtheria Toxin-Antitoxin Immunization

During the last year the Department of Health of the city of New
York has placed at the disposal of the profession the aforementioned
product for the purpose of effecting permanent immunity in persons
susceptible to diphtheria as demonstrable by Schick's reaction.

"The usual injection for all ages is approximately 400 times the
fatal dose† for a half-grown guinea pig, to which has been added just

---

†A. Zingher (Jour. Am. Med. Assn., Nov. 13, 1920) found that quite a number of Schick
test-outfits furnished by commercial laboratories do not contain a sufficient amount of toxin.

sufficient antitoxin to neutralize it. This is about four units of anti-toxin. The injection usually contains 1 c.c. of fluid and is made sub-cutaneously. The mixture is tested very carefully for its harmlessness before being used, and if so tested is absolutely safe. As it ages, the toxin disappears more rapidly than the antitoxin. A second and third injection of the same amount made at weekly intervals add greatly to the quantity of the antitoxin development from the first injection.

The Local and Constitutional Reaction

"The diphtheria toxin-antitoxin mixture contains besides the neutral-ized toxin a considerable amount of protein substance. This is partly formed of the proteins originally present in the broth in which the bacilli were grown and partly from the remains of broken down or di-gested bacilli in the cultures. The reaction to the protein in the injec-tion is similar to the reaction to the typhoid vaccine but it is of less severity.

"The element of age is very important. The infant shows in the great majority of cases neither a local nor a constitutional reaction, while grown up children and adults exhibit in perhaps 30 per cent of the cases considerable local swelling and more or less definite constitutional disturbance. Within 24 to 72 hours all disturbance is over. No lasting deleterious results have occcurred. Children of ages between one and ten years vary in the amount of reaction according to their age. The youngest shows the least and the oldest the most.

The Immunization Response in Susceptible Children

"Those persons who are naturally immune against diphtheria are usually so from having antitoxin, but may be so from the possession of other protective substances. The antitoxin we can measure by the Schick test, but we have no practical way to detect bactericidal substances.

The Immunizing Results

"These are measured by the percentage of susceptible persons who be-come immune, and by the persistence of the immunity. The antitoxin de-velops slowly after the injections are begun and gradually increases. In only a few cases does an appreciable amount of antitoxin develop in less than three weeks after the first injection. The majority respond during the second month. There are a few who become fully immune only dur-ing the sixth month. The results in 529 children who were carefully ob-served were as follows:

Number of Doses of 1 c.c. Toxin-Antitoxin	No. of Children	No. of Children Immune Three Months after Injection	Per cent Immune after Three Months
1	239	175	73
2	89	80	90
3	201	191	95

"These figures approximately agree with our results in thousands of cases. In young infants who are still retaining their parents' antitoxin, transferred to them passively before birth, we have less successful results. Tested one year afterwards only about fifty per cent were found to be immune. This percentage is about twice as great as among those not treated. Some 2,400 infants of an age under one week have been injected with absolutely no bad effect." Similar observations were made by J. Blum in an Infant Asylum accommodating 1,076 children.*

"The inmates of two institutions in New Jersey have been Schick tested for the fifth time for immunity to diphtheria, after one active immunization with toxin-antitoxin. The children were found to have remained immune from four to four and a half years. This is the longest period over which such tests have been made, so far as is known. The Leake and Watts Home, in New York, was also tested and the few children remaining in the home, since the first test four years ago, were found to be still immune.

"The 4,500 inmates of the State Insane Asylum at Kings Park have been Schick tested for immunity to diphtheria, and all patients showing susceptibility immunized by toxin-antitoxin.

"The children at the Colored Orphan Asylum at Riverdale-on-the-Hudson, who had previously received toxin-antitoxin, have been retested by the Schick test. The result shows that 104, out of the 111 children injected, have developed active immunity to diphtheria."

Antitetanic Serum

Like diphtheria antitoxin, antitetanic serum is obtained from the blood of horses previously immunized to the toxin of the tetanus bacillus. Its efficacy as a curative remedy is as yet awaiting indisputable demonstration, but its value as a preventive of tetanus is authoritatively established. Whenever there is reason to fear tetanus infection (e. g., contused or lacerated wounds—toy-pistol wounds—soiled with earth or other foreign matter) especially when an unusually large number of tetanus cases prevail, it is imperative promptly to administer tetanus antitoxin as a prophylactic measure.

*Active Immunization Against Diphtheria in a Large Child-Caring Institution, Am. Jour. Dis. Child. July, 1920.

Tetanus antitoxin is usually administered intraspinally, intravenously, and subcutaneously in doses of 1,000 to 1,500 units; the dose is repeated as a preventive measure after ten days, as a curative (3,000 to 5,000 units) once a day. (See p. 227.)

Antimeningitis Serum (Flexner)

This serum acts specifically in cerebrospinal meningitis due to the diplococcus intracellularis (Weichselbaum) only. If used by the subdural and intravenous methods in suitable doses, promptly and at proper intervals, it is capable of greatly diminishing the fatality generally due to the disease; of reducing the period of illness, and, in a large measure, of preventing the chronic lesions and types of the affection.

After reducing the intracerebrospinal pressure by withdrawal, by lumbar puncture (Fig. 175), of about 30 to 60 c.c. of cerebrospinal fluid, we inject 30 c.c. of the serum into the spinal canal by means of an antitoxin syringe or by gravity through a funnel and rubber tube attached to the puncture needle. The modern serum containers greatly facilitate the administration of the serum. The injection is repeated daily for three or four days or longer until the diplococci disappear. In fulminating cases a second dose may be given after the lapse of twelve hours. If after a period of apparent recovery the symptoms recur and the diplococci reappear, the injection should be repeated. The serum is practically useless in cerebrospinal meningitis after the condition of hydrocephalus has supervened.

"Up to a short time before the war began a single type of meningococcus was generally accepted as the cause of epidemic meningitis. Dopter[1] was the first to classify meningococcus-like organisms into distinct types. In connection with a recent very lucid description of the manner in which the types of meningococcus came to be recognized, he[1] has described in detail the modifications which have resulted in the treatment of meningitis. Four types of meningococci are now generally recognized, designated as Types A, B, C and D. Type A appears to have been the common one before the war, being found according to Dopter in from 95 to 96 per cent of the cases. Of the other types, sometimes called parameningococci, B is most common, C and D exceptional. Infections by Type B increased during the first two years of the war, and at the end of 1917 about 50 per cent of the cases in the French army were of this form. Each of these various types of organism is affected only by its own specific serum. A case of meningitis caused by the Type B Meningo-

[1]Dopter, C.: Recent Work on Cerebrospinal Fever, Lancet, French Supplement, 1:1075. (June 21) 1919.

coccus is not influenced by a serum prepared from Type A organisms. Consequently it has been necessary to prepare serums from each type of organism for use in the treatment of meningitis due to the corresponding type.

"For the most efficient serum treatment of epidemic meningitis, two things now appear essential: (1) an accurate biologic determination of the type of organism concerned in the individual case, and (2) the administration of the serum prepared from the corresponding type. Doptor is not in favor of using polyvalent serum except as a measure of precaution until the laboratory examination has determined the type present in the case. As soon as the type is known, the corresponding monovalent serum should be substituted. He believes that 'too much polyvalency might conceivably involve risk of diminished potency.' Those who have treated meningitis with serum have observed that occasionally cases occur which are not appreciably benefited by the polyvalent serum used, and in such cases the spinal fluid does not clear up neither do the meningococci decrease in the fluid, as is usual in most cases. Sometimes another make of polyvalent serum may be active, and it is advised to make use of this expedient, with the hope that a strain of meningococcus corresponding to the one causing the infection may have been among those employed in preparing the serum. At best this is not satisfactory. It is much to be desired that serums should be prepared from the several types so that they may be available for cases which do not respond promptly to the polyvalent serums. Accurate differentiation of the type of infecting organism by biologic tests is essential before the treatment can be carried out with a high degree of precision. It has been found that the cases prevailing in a group of individuals both in the meningeal exudate and in the nasopharynx of carriers are usually of one type. Mathers and Herrold found that, in a camp near Chicago, almost 86 per cent of the cases of meningitis were due to Type A (Group 1), and in the city of Chicago at the same time more than 86 per cent of the cases were due to Type B (Group 2). If investigation revealed the prevalence of one type in a community or epidemic, a serum high in immune bodies for that type would be reasonably used for routine treatment if it were not feasible to make a biologic differentiation in each case. As the abnormal conditions of army camps disappear, it will be of interest to note whether the prevailing type of meningococcus will again be the Type A, as was apparently the case before the war. Study of cases of epidemic meningitis has served also to emphasize the fact that the meningococci are found not only in the meninges, but also often in the blood, joints, etc. When serum is injected intraspinally, it rapidly passes into the circulation; but it is desirable to secure a greater concentration of anti-

bodies in the blood than is secured in this way. This can be brought about by intravenous or intramuscular injection of serum. It would probably be a useful practice to combine intramuscular with intraspinal injection in all cases. When intravenous injections are used, all precautions to avoid anaphylactic shock should be taken." (Jour. Am. Med. Assn., Oct. 11, 1919.)

With the demonstration of the meningococcus in the blood, several clinicians have recently begun to administer antimeningococcus serum intravenously as well as intraspinally. The intravenous method is recommended especially in severe cases. Major W. W. Herrick, who has had under observation 265 cases of epidemic cerebrospinal meningitis, at Camp Jackson, and has employed this method with a great reduction in the ordinary mortality in this affection, offers the following suggestions which relate, of course, to adults, but can readily be modified to suit the needs of children:

Important Points in Intravenous Serum Treatment.—It must be employed with boldness, yet with care. One must be prepared to give from four to eight massive injections by vein of from 80 to 150 c.c. during the acute stages of the disease or a period of from two to four days. There is much more danger in insufficient than in excessive intravenous serum administration. He has, in fact, in 128 cases so treated, had no serious serum effects. His regrets have been that serum was not more freely used in many of the early cases.

The desensitization by subcutaneous injection of 1 c.c. of serum one hour before the introduction of serum into the vein and the cautious injection of the first 15 c.c. at the rate of 1 c.c. per minute are the secrets of safe intravenous serum therapy. Immediate stopping of the injection with the appearance of dyspnea, pallor, cyanosis, vomiting, weak, rapid or irregular pulse or other immediate serum effects is essential. Renewal of the attempt after two or three hours is rarely unsuccessful. Even those patients thoroughly sensitized to serum by earlier courses of treatment can be treated safely with these precautions.

In those prolonged cases in which meningococci persist in the spinal fluid and in which the patients are made uncomfortable by intraspinal injections, showing increased opisthotonos, and severe pain in the head or back or lower extremities following the treatment, it is better to omit all interference. At times drainage may be necessary with or without further intravenous injections. Many of these prolonged cases apparently become intolerant of intraspinal serum injections. If satisfactory response does not follow a series of eight or ten intraspinal treatments, it is generally best to cease injecting serum intra-

spinally, continuing drainage only if there is discomfort from increased intracranial pressure or apparent danger of blocking the foramina.

In relapsing cases the entire cycle of treatment must be repeated with the same thoroughness and care used in the initial course. The organism cultivated from the blood or spinal fluid may be used to determine the presence of agglutinins in the serum employed. Valuable evidence can thus be obtained as to the specificity of the serum for the strain of meningococcus present. This is of the highest importance. Therapeutic results seem to parallel the agglutinin content of the serum for the special strain of meningococcus involved. Commercial serums are frequently lacking in high agglutinin content, and their therapeutic effect is often disappointing. In the absence of desirable results from a given serum, use should promptly be made of serum from another source. This may be of vital moment to the patient. The standardizations of serums by governmental authority is an urgent need.

Of course, the dosage varies with the age of the patient. A third of the adult dose ought ordinarily to suffice for children under five years of age, and one-half of the adult dose for older children. In young infants the longitudinal sinus route may be used for the injection of the serum; in older children the basilic vein.

Several other sera (e. g., antipneumococci, antidysenteric) are now on the market. Their curative merits, however, are still unestablished.

Bacterial Vaccines

Following upon the great researches of our contemporary pathologists, bacteriologists and clinicians, A. E. Wright, of London, has demonstrated the remarkable fact that emulsions of dead bacteria—bacterial vaccines so called—if injected subcutaneously increase chemotaxis and, therefore, phagocytosis. The molecular group produced by the presence of the killed bacteria in the blood that renders the living bacteria of the same species a ready prey to the phagocytes he designated "opsonin," corresponding to the Greek verb "opsono" —I cater for, I prepare victuals for. He also devised a method to determine the "opsonic index," of sensitizing power of the blood, so that in a given case of infection one can, as it were, measure the opsonin content of the blood and increase it, if found below par.

Bacterial vaccine therapy is mostly limited to local infections, e. g., furunculosis, phlegmons, carbuncles, where the offending microorganisms can readily be determined by microscopic examination of the discharges, and accordingly the vaccine chosen to meet the indications.

Of the numerous vaccines thus far recommended, the staphylococcus

and streptococcus vaccines have actually stood the test and proved of great utility. They are deserving of more general application.

Favorable results are also on record from the use of vaccines prepared from the bacillus coli (in colicystitis); from gonococci (in gonorrheal affections, especially vulvovaginitis); from typhoid bacilli (in typhoid, especially as a preventive measure) and from combined pertussis vaccine (as a preventive, and in the early stages of whooping-cough as a therapeutic measure. See ''Pertussis'').

The inoculations are given by means of a sterile hypodermic syringe, in the same manner as antitoxin. In children particularly it is advisable to begin with small doses, let us say, 50 million staphylococci, or 2 million streptococci, and to increase the dose of each succeeding injection.

In order to obtain prompt results it is essential to know not only the specific infecting microorganism but also its variety; for instance, whether the offending staphylococcus is an aureus, albus, or citreus, since the employment of a different variety of vaccine is apt to prove useless. For Influenza Vaccines see p. 354.

Bacterial vaccines are often prepared directly from cultures obtained from the individual to be treated—autogenous vaccine.

Tuberculin Tests and Tuberculins

These bacterial products are invaluable in the early diagnosis of tuberculosis in children. By means of tuberculin we are enabled to detect from 90 to 95 per cent of cases of tuberculosis, often at a time when no other clinical manifestations or bacteriologic examinations indicate its presence. It has furthermore the great advantage that its use calls for no complicated procedures, methods, calculations or instruments. According to von Pirquet, the specific test is based upon the fact that an individual contracting tuberculosis develops a hypersensitiveness of the tissues (so-called ''allergia'') to the poison of tubercle bacilli which is manifested by a local inflammation or systemic disturbance.

The tuberculin reaction may be elicited in the following manner:—

1. *The Cutaneous Method (von Pirquet).*—After cleansing the anterior surface of the forearm with soap, water and ether, two small abrasions (as for vaccination) or punctures of the skin are made at an interspace of about 2 inches. On one of the two abraded spots a drop of a 50 to 100 per cent solution of old TB is applied and allowed to dry. If tuberculosis is present, a red pea- to bean-sized papule appears after from twenty-four to forty-eight hours at the point of contact of the

injured skin and tuberculin, while the other nontuberculized spot remains free from the inflammatory reaction.

2. *Conjunctival Method (Calmette).*—A drop of ½ to 1 per cent (trying the weaker solution first) of old TB solution is instilled into the conjunctival sac of one eye. In the presence of tuberculosis a positive reaction is manifested within twenty-four hours by reddening of the caruncles and semilunar fold of the conjunctiva and injection of the corneal conjunctiva. The other eye remains normal.

3. *Nasal Method (Wolff-Eisner and Calmette).*—A cotton tampon saturated with a 1 per cent TB solution is applied against the nasal septum and allowed to remain there for about ten minutes. In from eighteen to forty-eight hours a peculiar exudation appears which dries and forms a yellow crust upon a congested mucosa. From this clumps of extravasated red cells project here and there as minute reddish points. The crust generally falls off in from four to six days.

4. *Percutaneous Method (Moro).*—This method is less reliable than the aforementioned procedures. A 50 per cent tuberculin ointment is rubbed over about a square inch of epidermis until absorbed. If the reaction is positive, papules appear within from twenty-four to forty-eight hours.

5. *Subcutaneous Method.*—Very rarely employed in young children.

Tuberculin Therapy.—Tuberculin treatment, like so many similar new, in their therapeutic effects grossly inflated, remedial measures, has for several years been relegated into oblivion. Yet tuberculin, properly employed and in suitable cases is of remarkable benefit in tuberculous affections, more particularly in those of the small bones, joints, glands and skin. Its curative action is due to stimulation of defensive powers of the body and its resistance to the pathogenic action of the tubercle bacillus and its toxin. If these means of defense are not in a condition to be favorably influenced by the tuberculin, the therapeutic results, of course, will be *nil.* Hence the importance of beginning the treatment as soon as tuberculosis is diagnosed or even suspected (E. Béraneck). Furthermore, the important thing is to begin with a small dose of a very dilute solution, and to continue to inject (twice a week) three or four times at least before arriving at any definite estimate of the need of a larger dose. If the effect seems favorable, the same dosage should be continued for weeks or months, so long as the patient is deriving benefit from the treatment. On the other hand, if three or four injections of the initial small dose seem to exert no beneficial effect, a somewhat larger dose is administered and its therapeutic action carefully observed in the same manner as with the smaller dose. The initial dose of the tuberculin solutions, presently

to be enumerated, should be one-millionth of a milligram in non-febrile cases and a smaller dose in those showing moderate fever. The subcutaneous injection is made with the usual aseptic precautions. If the injection is followed by marked systemic disturbance and high fever, the treatment is temporarily discontinued and a smaller dose begun with, after the fever has subsided.

The tuberculin (Koch) is diluted with sterile physiologic salt solution or ½ per cent carbolic acid water in the following manner:

Sol. No. 1	Tuberculin	1 c.c.	1 c.c.	0.1
	Diluent	9 c.c.		
Sol. No. 2	Sol. No. 1	1 c.c.	1 c.c.	0.01
	Diluent	9 c.c.		
Sol. No. 3	Sol. No. 2	1 c.c.	1 c.c.	0.001
	Diluent	9 c.c.		
Sol. No. 4	Sol. No. 3	1 c.c.	1 c.c.	0.0001
	Diluent	9 c.c.		
Sol. No. 5	Sol. No. 4	1 c.c.	1 c.c.	0.00001
	Diluent	9 c.c.		
Sol. No. 6	Sol. No. 5	1 c.c.	1 c.c.	0.000001
	Diluent	9 c.c.		
Sol. No. 7	Sol. No. 6	1 c.c.	1 c.c.	0.0000001
	Diluent	9 c.c.		
Sol. No. 8	Sol. No. 7	1 c.c.	1 c.c.	0.00000001
	Diluent	9 c.c.		
Sol. No. 9	Sol. No. 8	1 c.c.	1 c.c.	0.000000001

Complement-Fixation Reaction in Tuberculosis

During the last ten years considerable progress has been made in the detection of tuberculosis by the aforementioned reaction. The technic is the same as in the Wassermann reaction (q. v.) except for the antigen, which consists of an emulsion of ground tubercular bacillary bodies. The longer the bacilli are ground, the better the antigen. It matters but little whether the bacilli are triturated dry (Miller), wet (Fleischer), by boiling in glycerin (Petroff) by dissolving off the wax and suspending (Cooke, Wilson), or allowing it to occur by lysis (Corper). From a careful experience with 6500 tests, W. W. Watkins and C. N. Boynton* have recently formulated the following conclusions:

The Miller antigen is serviceable, practical and efficient for the complement-fixation test in tuberculosis.

The reaction is specific for tuberculosis and, when positive, should be interpreted as indicating tuberculosis of some degree of activity. When the Wassermann and tuberculosis fixation reactions are both positive, they should be interpreted without relation to each other.

The positive fixation reaction can be interpreted as indicating tuber-

---

*Jour. Am. Med. Assn., Oct. 2, 1926.

culosis, either active at the time, or recently active. The focus may or may not be of clinical significance, which fact must be determined by other means.

The negative fixation reaction indicates either absence of infection, excessive activity of the disease, exhausting the antibody, or arrest of the disease with spontaneous disappearance of antibody no longer required.

Serum Diagnosis of Syphilis (Wassermann)

The substances employed in this reaction are as follows: (1) Complement. One to ten dilution of fresh guinea pig serum in normal (0.85 per cent) salt solution. (2) Antigen. Alcoholic extract of a syphilitic organ or suspension of an organ in weak carbolic acid solution (1 per cent). (3) Amboceptor. Inactivated serum of rabbit which has been highly immunized against sheep red-cell by five or six injections of increasing amounts of sheep red-cells. The amboceptor is standardized by putting in each of a series of test tubes 1 c.c. of complement and 1 c.c. of 5 per cent emulsion of sheep red-cells. Different amounts of the inactivated rabbit serum are added to the tubes, beginning with 0.01 c.c. to 0.1 c.c. The tubes are then incubated one hour. That in which complete hemolysis occurs contains just enough of amboceptor to dissolve 1 c.c. of 5 per cent emulsion of sheep red-cells. Double the quantity is the amboceptor to be used. Suspected serum to be used is drawn from a superficial vein with a medium-sized exploratory needle under strict aseptic precautions, about 5 c.c. being sufficient. The blood is centrifuged and the cleared serum inactivated by heat for thirty minutes at 56° C.

Test.—Put 1 c.c. of complement, 2 drops of suspected serum, about 0.1 c.c. of antigen in test tube and incubate one hour at 37° C. Then add the amount of amboceptor, determined by standardization, and 1 c.c. of 5 per cent emulsion of sheep's red-cells suspended in normal salt solution and incubate again for one hour. Then place in ice box for six hours. Complete hemolysis is indicated by a clear burgundy-red solution, showing no precipitate. No hemolysis is indicated by a solid, opaque sediment of the unaffected sheep cells at the bottom of the tube, while the supernatant fluid is clear and colorless.

Result: Hemolysis, no syphilis; syphilis, no hemolysis. The control test is the same except that the antigen is omitted. (After G. M. Gould and R. J. E. Scott.)

The Noguchi method of the serum diagnosis of syphilis is a modification of the Wassermann reaction. "(1) He prepares the antigen by extracting a lipoid substance from the liver and heart of dogs and cows.

(2) Instead of using sheep's corpuscles in the hemolytic series, he employs human corpuscles, owing to the fact that a certain percentage of human sera tested produced hemolysis of the sheep's corpuscles. (3) In his test, therefore, he obtains the hemolytic amboceptor by immunizing rabbits with washed normal human corpuslces. (4) Another important improvement in the technic is the preservation of the specific antigen and the hemolytic amboceptor, which rapidly lose their strength in solution, in a dried form by soaking measured strips of filter-paper (.5 mm. square) with each. His test is carried out as follows: A strip of antigen filter-paper is brought in contact with a definite quantity of the human serum to be tested and fresh guinea-pig's serum added, the whole being suspended in isotonic salt solution. This is allowed to stand at incubator temperature and then the hemolytic series added by taking a strip of the hemolytic amboceptor paper and a definite quantity of washed normal human blood corpuscles."—(Tyson's *Practice of Medicine.*)

Serum Diagnosis of Typhoid

(GRUBER-WIDAL)

The blood of persons suffering from typhoid, when added to a broth culture of typhoid bacilli, arrests the characteristic movements of these germs and produces their agglutination and sedimentation.
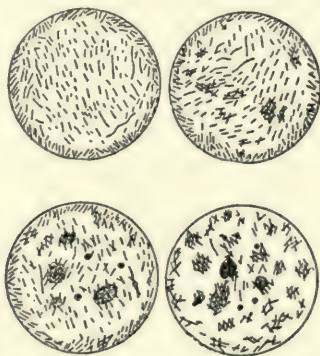
Fig. 5.—Stages in Widal reaction of typhoid (after Robin).

This phenomenon may be observed macroscopically in a suspension of bacteria in test tubes; or microscopically when the bacteria are mixed with the blood and mounted in a hanging drop preparation. The test is generally positive in typhoid patients after the fifth day of the disease and several weeks thereafter.

The blood (or serum from a blister) is obtained from the skin covering the ear lobe. After cleaning this part, the lobe is pricked with

a sterile needle, and two drops of blood are placed on a glass slide, one near each end, and allowed to dry in the air. The examination can then be undertaken any time thereafter by diluting one drop of the blood in ten or twenty parts of the typhoid culture.

Weil-Felix Reaction of Typhus Fever

What is known as the Weil-Felix reaction has recently come into use abroad in the diagnosis of typhus fever, and as its value has seemingly been proved, it should be employed for confirmation of clinical diagnosis in all suspected cases of typhus or continued fever.

This reaction is similar to the Widal test in typhoid, and consists in testing the agglutinating power of the patient's blood serum on a suspension of bacilli obtained from cases of typhus fever, which have tentatively been classed as various members of the Proteus group of organisms. The bacilli in question were described by Weil and Felix as short Gram-negative rods, slightly motile, forming blue colonies on Conradi-Drigalski medium, and colonies which become pink on Endo medium. The organisms, according to these authorities, ferment dextrose and curdle milk, with the development of an acid reaction, and liquefy gelatin. They are also stated to produce indol.

The technic of the agglutination test (as recommended by Weil and Felix) is as follows: The growth of the proteus-like bacillus on an agar-slant is suspended in a small quantity (2 c.c.) of 0.9 per cent salt solution, and this is mixed in the proportions of 1 to 25, and 1 to 50, with serum from the suspected case. Hanging drops of these dilutions are then examined microscopically after a half hour's incubation at 37° C.

In positive cases, agglutination should take place in dilutions of 1 to 25, on the 6th day; and, by the 12th day of the disease, in dilutions of as great as 1 to 200, or higher.

Allergy- or Food Idiosyncrasy-Test

This test is of great diagnostic and hence therapeutic value in determining food idiosyncrasies which are not rarely responsible for marked gastrointestinal disturbances, skin affections and asthma.

The technic is the same as in von Pirquet tuberculin test, using the soluble food product instead of the tuberculin.

A positive reaction is manifested in from ten to thirty minutes by the appearance of a blotchy papular eruption about ½ inch in diameter, accompanied by local or general itching of the body. This may be followed by asthmatic breathing. A positive reaction, of course,

calls for the removal of that particular kind of food from the dietary, until the susceptibility has disappeared.

The following food-testing-products are marketed by the Arlington Chemical Company, Yonkers, N. Y., or Squibb and Sons, New York.

Almond	Lactalbumin
Banana	Lamb
Barley	Lentil
Bean	Lettuce
Beef	Lobster
Buckwheat	Oats
Cabbage	Onion
Carrot	Orange
Casein	Oyster
Clam	Pea
Cocoa	Peanut
Codfish	Pork
Corn	Potato
Crab	Rice
Cucumber	Rye
Egg Albumin	Squash
English Walnut	Strawberry
Grapefruit	Tomato
Haddock	Wheat

IV. MATERIA MEDICA AND THERAPEUTICS

(Including Hydrotherapy, Electricity, Massage, and Climatotherapy)

No one method of treatment suits all cases. Some diseases subside spontaneously, if let alone; others go from bad to worse if not treated promptly and energetically. Some affections yield readily to biologic remedies, others to crude drugs or synthetic pharmaceutical preparations, and again others respond to change of climate, mode of living and eating, and to remedial measures other than pharmaceutical, such as hydrotherapy, massage, electricity and the like.

Our duty being to alleviate suffering, we owe it to our patients to keep pace with the advances of the time and to employ every useful method of treatment regardless of its source or character. "The period of exclusiveness is past." While a certain degree of conservatism is always wise and safe, skepticism to well-tried remedies is worse than folly.

Hydrotherapy

The virtue of water as a therapeutic agent varies with the idiosyncrasy of the patient, the temperature of the water employed and the method of its application.

Heat applied to the surface of the body produces a relaxation of the vasomotor system. The cutaneous vessels dilate and become more ac-

tive, diaphoresis ensues, and effete matter is eliminated. The volume of blood in the deeper structures is diminished; hence, congestion relieved. The temperature of the body is first increased, but after free diaphoresis considerably lowered.

Cold contracts the terminal blood vessels and stimulates the internal circulation. It reduces the temperature of the body not only by conduction but also by inhibition of heat production. Soon after discontinuance of the cold a reaction takes place, respiration becomes deep and full, more carbon dioxide is excreted and the supply of oxygen is increased. The pulse, which is at first feeble, soon becomes full and strong; the chilliness and rigor disappear, and a sensation of warmth pervades the body surface. The blood current in the capillaries becomes gradually accelerated and the internal circulation relieved of its tension.

The External Use of Water.—Neither extreme heat nor extreme cold should be employed in the treatment of diseases of children. Heat should be avoided on account of the severe depression, and cold because of the shock it is apt to produce.

Cold Sponging.—In the employment of cold water in the treatment of diseases of children, sponging advantageously supplants the cold bath. The temperature of the water should vary between 70° and 90° F. Three basins of water, one each of 70° F., 80° F. and 90° F., respectively, are placed at the bedside. The child is stripped and laid upon a blanket, and by means of cloths the surface of the body is sponged for from two to three minutes, in the following order of succession: face, neck, chest, back, abdomen, buttocks, upper and lower extremities. The warmest water (90° F.) is used first and the coldest (70° F.) last. Each part of the body should be thoroughly dried immediately after it has been sponged. The indications for the use of the sponge bath are hyperpyrexia and nervous irritability; constitutional disorders, such as anemia, chlorosis, scrofula, etc., and in cases in which a general tonic effect is desired. In the latter conditions sponging should be followed by active friction.

Cold Wet Pack.—The child is stripped and blankets are placed over and under it. A small sheet is dipped in water at a temperature of 70° to 90° F., thoroughly wrung out and wrapped loosely around the patient. The child's body is then enveloped in the blankets. To reduce high temperatures, for example, in typhoid or pneumonia, ice may be rubbed over the pack. The next pack is applied after an interval of ten minutes and may be repeated from ten to twelve times in twenty-four hours. The feet should be kept warm by artificial heat.

Vapor Pack.—If the cold wet pack is allowed to remain in position for from one to two hours and loss of body heat prevented by thoroughly

covering the child with woolen blankets, the cold pack is converted into a warm pack which produces effects similar to those obtained from a vapor bath—namely, free diaphoresis, lowered activity of the nervous system, calm and repose, and equalization of the internal circulation. The vapor pack is, therefore, invaluable in acute catarrhal conditions of the air passages, in nephritis, dropsical effusions, muscular rheumatism, eclampsia, hyperesthesias, etc.

Wet Local Compresses (Priessnitz).—Cold Compresses.—These are applied in all forms of local inflammation, to relieve pain, swelling, heat and redness. In order to obtain good results, the temperature of the water should vary between 50° and 60° F., and the compress left in place and kept cold either by frequently sprinkling cold water over it or by the application of an ice bag.

Indications: Headache, angina, acute pharyngitis and laryngitis, hemoptysis, appendicitis, intestinal hemorrhage, etc.

Warm Compresses.—While cold compresses delay the flow of blood and cell activity, warm compresses accelerate the blood-current and promote cell activity. They are applied by means of cloths immersed in water at a temperature of about 100° F., thoroughly wrung out, then covered with flannel and rubber tissue or oiled silk to prevent rapid evaporation and cooling. The compresses should be changed as soon as they become dry.

Indications: Neuralgia of the head; throat affections after subsidence of the acute inflammatory stage, to promote absorption of diseased products; in exudative pleuritis; in bronchitis, to allay severe cough and to promote expectoration; in all spasmodic conditions of the intestines; to hasten suppuration and relieve stasis.

Baths.—Tepid Bath.—This is a very useful bath in children. The temperature of the tepid bath varies between 85° F. and 92° F. It is employed in diseased conditions requiring soothing, for example, in eruptive skin diseases and as an antipyretic in infectious diseases.

Warm Bath.—In a general sense, this is the most valuable bath in the treatment of diseases of children. It tranquilizes the nervous system, equalizes the circulation, produces diaphoresis and reduces temperature.

Indications: All spasmodic conditions; affections of the lungs and kidneys; exanthematous diseases, and nervous affections, such as hysteria, etc. The temperature of the bath should vary between 92° F. and 98° F. The patient should remain in the bath for from two to five minutes. The warm bath is sometimes employed as a *permanent bath,* in extensive burns and wounds, and in skin diseases associated with intense itching. The patient is suspended in the bath on a sheet.

The water is kept at an equal temperature by proper arrangement of inflow and outflow.

Hot Bath.—The temperature of the hot bath may be carried gradually as high as 108° F., and the patient should remain in the bath for from one to three minutes. It is very useful in collapse, convulsions and chronic rheumatic conditions. It is occasionally administered to break up a "cold," and to produce rapid diaphoresis. While in the bath the patient's head should be kept cool by an ice bag.

Shower Bath.—Cold shower baths are generally given for their stimulating effect. Hence, they are of great value in nervous affections, such as neurasthenia; in enuresis, and as a general tonic. For these purposes one shower (shock) at a time is sufficient. The shower bath should be followed by active friction.

Aspersion Bath.—The value of cold water dashed suddenly over the frame or directed in a steady, broad stream upon some particular part, is very great. The cases in which such a mode of treatment is beneficial are numerous. The following are a few of the more important: Where the muscular power of a leg or arm is impaired from long inaction, as in cases of fracture, dislocation, sprains and partial paralysis. The patient sits in a bath tub or on the floor and the operator, standing on a table, directs the stream of cold water upon the affected part from a watering can from which the sprinkler has been removed. This mode of treatment is rendered particularly serviceable if the circulation is quickly restored by vigorous dry friction for several minutes. It is also efficacious in systemic poisoning from drugs, suffocation from noxious gases, etc.

Medicated Baths.—Aside from the natural mineral baths obtained in the celebrated spas, which will be discussed later, a number of artificial baths are commonly used in the treatment of diseases of infancy and childhood. The efficacy of these baths is, in the majority of instances, probably due to the effects of heat or cold and friction employed with the nonmedicated bath.

Nauheim Baths.—These baths are used chiefly in the treatment of chronic heart disease, and diverse neuroses. Where natural springs are not within reach, the baths may be prepared by the addition of the following ingredients which evolve carbonic acid gas; the therapeutic action depends chiefly upon its stimulating effect upon the skin.

Sodium Chloride	4	lbs.
Sodium Bicarbonate	½	"
Calcium Chloride	4	"
Hydrochloric Acid	1	"

The hydrochloric acid is added gradually after the other ingredients have been thoroughly dissolved in the bath. The baths should be taken two or three days in succession, followed by a respite of two days.

Aromatic Bath.—About six ounces each of chamomile flowers, calamus roots and peppermint leaves are tied up in a muslin bag and thrown into a warm bath. Aromatic baths are recommended in marasmus, infantile, spinal and other forms of paralysis, in sclerema, etc.

Bran Bath.—Two or three pounds of wheat bran are boiled for about an hour in about three quarts of water. The decanted liquid is added to the bath. It is useful in intertrigo, eczema, pemphigus, lichen, strophulus, etc.

Malt Bath.—A few ounces of malt extract are added to the bath. Malt baths are recommended in rachitis, spasm of the glottis, and in general debility.

Mercurial Bath.—This form of bath is employed as an adjuvant in the treatment of syphilis. It is usually prepared by the addition of 20 to 30 grains of calomel, or 0.5 to 1.0 grams (7 to 15 grains) of bichloride of mercury.

Mustard Bath.—Two or 3 ounces of mustard are dissolved in a few pints of tepid water and added to the bath, or the mustard powder is tied up in a bag and thrown in the tub. The temperature of the bath may vary between 100° F. to 106° F. It may be administered in the form of a sitz bath or full bath. The patient should remain in the bath for from three to ten minutes. Mustard baths are indicated in collapse, shock or heart failure from any cause, in sudden congestion of the lungs or brain, etc.

Sea Salt Bath.—About 2 pounds of sea salt are dissolved in the bath of 4 or 5 gallons of water. It is stimulating in its effects, and useful in rachitis, various forms of paralysis, etc.

Soap Bath.—This form of bath is employed in the treatment of prurigo, lichen, strophulus, scabies, etc. It is prepared by the addition of from 3 to 6 ounces of soft green soap to 5 gallons of water.

Sulphur Bath.—Half to one ounce of potassium sulphuret should be added to each bath. In some cases the addition of about 3 ounces of animal gelatin is of advantage. Sulphur baths are deserving of recommendation in rheumatism, eczema, prurigo, urticaria, lead poisoning, etc.

The Internal Use of Water

The benefits derived from the internal use of water are manifold, but unfortunately greatly underestimated. Water taken by the mouth in moderate quantities—large amounts weaken diges-

tion—cleanses the alimentary canal, stimulates peristalsis and produces diuresis and diaphoresis. To a certain extent it acts also as a food. In acute diseases associated with anorexia the free use of water will often sustain life for weeks. In febrile diseases water not only quenches thirst, but aids also in the reduction of temperature. Water stimulates expectoration, and in the form of cracked ice checks vomiting. For the latter purpose small sips of hot water are sometimes resorted to.

Lavage.—Stomach washing in children is performed in the same manner as in adults. Its field of usefulness, however, is much wider. It is invaluable in cases of acute, simple and toxic gastritis, pyloric stenosis, cholera infantum, chronic indigestion and difficult feeding. A funnel with a few feet of rubber tubing, to which a soft rubber catheter (No. 12 or 14) is joined by means of a glass cannula, is the best apparatus for stomach washing. About 10 inches of the catheter should be passed beyond the lips. The temperature of the irrigating solution should be about 100° F., or higher, if special indications arise. The quantity of solution to be instilled varies with the capacity of the child's stomach. Generally, pure, boiled water answers all medicinal purposes, except in poisoning, in which instance antidotes may be employed. In hyperacidity of the stomach bicarbonate of soda or lime water may be added. Lavage is contraindicated in heart disease and hemorrhagic diathesis.

Irrigations.—The action of irrigations is chiefly mechanical. They are indispensable in the treatment of divers affections of the lining membranes of internal cavities. In chronic cystitis, for example, washing of the *bladder* by means of sterile or medicated (boric acid, silver nitrate) water will often rapidly effect a cure.

Irrigations of the *vagina* are frequently employed in vulvovaginitis. A slow current of water should be employed, permitting the fluid to return without injury to the adjacent parts. A fountain syringe with a small, sterile, soft rubber catheter attached, generally suffices for ordinary purposes. The water bag should be suspended about 2 feet above the child's body.

Irrigations with warm, sterile water are very beneficial in *ear affections,* such as impacted cerumen, foreign bodies in the external auditory meatus and in otitis media.

In febrile diseases, adenoids, chronic pharyngitis, etc., instillations of weak salt water or ichthyol solutions prevent and cure affections of the nasopharynx and ear; it often also relieves reflex cough and embarrassed respiration. Instillation may be performed by means of a teaspoon or dropper, and should be repeated at least twice a day.

Copious irrigations of the *mouth* with sterile or medicated (silver ni-trate, hydrogen peroxide) water are invaluable in the treatment of grave forms of stomatitis.

Enteroclysis.—The indications for *low enemas* are too well known to need further discussion. It may be mentioned, however, that in habit-ual constipation only small quantities of water should be injected into the bowel. Large quantities are apt to produce atony of the colon by overdistention and thus aggravate the disease.

High enemas are given by means of a flexible (colon) tube and a foun-tain syringe. High enemas not only remove effete material from the intestines, but by using water at a temperature of 80° to 90° F. also reduce temperature. Hence, they combine two therapeutic measures, which are of signal benefit in all gastrointestinal disorders, peritonitis, typhoid, etc. Soap suds, bicarbonate of soda, turpentine, starch and salt, among other adjuvants, may be added according to indications.

Saline injections stimulate the kidneys and promote elimination of putrid material. They stimulate the circulation and supply the deficiency of body fluids in conditions associated with an excessive drain of fluids. Saline injections are, therefore, a sovereign remedy in uremia, typhoid fever, scarlet fever, smallpox, measles, diphtheria, eclampsia, anemia, hemorrhages, and in shock after surgical operations, etc.

A physiologic (0.9 per cent) salt water solution at a temperature of from 100° to 110° F. is generally used. It should be injected slowly through a colon tube, and continued for from fifteen to twenty minutes, or by Murphy-drip for several hours in succession.

Saline injections are contraindicated in chronic kidney disease, the salt acting as an irritant.

Hypodermoclysis.—Subcutaneous injection of salt water (110° F.) is performed by means of an ordinary fountain syringe with an antitoxin syringe needle attached. The syringe needle and skin should be ren-dered aseptic. The injection should be made in places where there is an abundance of subcutaneous cellular tissue; for example, the anterior surface of the abdomen and thorax. The current should be very slow, and the quantity of the saline solution to be injected should vary be-tween from 2 to 6 ounces, according to age and indications. Hypo-dermoclysis is of inestimable value in cases of collapse resulting from hemorrhage; in pneumonia; uremia; acute gastroenteritis with great loss of body fluids; and in leukemia. In infants it should be preferred to intravenous infusion. More recently good results have been reported from intravenous infusion by the longitudinal sinus route and by peri-toneal injection.

Intrasinus Injection.—In an infant with open fontanel this offers the best means of introducing fluid into the blood stream. The method was first studied by Tobler and was introduced in this country only a few years ago by Helmholz. By this technic, the fluid can be injected through the anterior fontanel directly into the superior longitudinal sinus. As the sinus lies from 2 to 5 mm. from the skin, it can be easily entered if the fontanel is not closed; at the posterior angle of the fontanel the sinus is wider and deeper. The child is held prone on the table by an assistant, while the needle is introduced in the median line just in front of the posterior angle. If the child is quiet, it is very easy to withdraw blood or to introduce fluid; by means of a Luer syringe, rubber tubing and a threeway cock any amount of fluid can be given without removing the syringe. The needle should be short, and the long point usually found on intravenous needles should be filed away. If a glass syringe is attached before introduction of the needle, constant suction may be maintained for the purpose of discerning when the sinus is entered. If negative pressure is not produced, blood will not flow so quickly, while the operator may push the needle through the inferior wall of the sinus, blood flowing only when the needle is withdrawn. This accident may also be avoided if the needle be introduced at an angle, directed backward.

Any solution adapted to intravenous administration can be given in this way; with physiologic sodium chlorid, glucose and other mild solutions there is practically no danger. It is also an excellent method for transfusion of citrated whole blood in infants.

In cases with a closed fontanel, the external jugular or femoral vein can often be used successfully.

Intraperitoneal Injection.—This was first used in St. Bartholomew's Hospital and was introduced in this country by Howland. As may be noted from the following observations of J. Aikman (Rochester, N. Y.)[*] its method of application is very simple.

The instruments needed are a medium-sized intravenous needle, an infusion bottle and rubber tubing. The skin of the abdomen is carefully sterilized with tincture of iodin and alcohol. The skin and subcutaneous tissue are picked up between the thumb and forefinger, and the needle is introduced in an upward direction through the abdominal wall in the midline just below the umbilicus. Care must be taken to avoid piercing a distended bladder, and while there is also danger of puncturing the intestine, no record of this accident has come to his attention. In cases in which necropsy was performed there was found a small hemor-

[*](Jour. Am. Med. Assn., lxxiv, No. 4, 1920.)

rhagic area in the abdominal wall and peritoneum, but no injury of serious importance.

When the needle has passed into the peritoneal cavity, the solution is introduced by gravity. At first he used a Luer syringe; but later he found it much easier to employ the infusion bottle. He has always used warm physiologic sodium chlorid solution, of which from 100 to 250 c.c., in older children from 300 to 400 c.c., may be given every twelve to twenty-four hours, in fact, if no untoward signs develop, fluid may be given until the abdomen becomes slightly distended. However, the injection must be made slowly in all cases, and overdistention of the abdomen must be avoided. After the operation, the abdomen is covered with a sterile dressing. It has been shown by the phenolsulphonephthalein test and by necropsy that from 40 to 60 per cent of the fluid is absorbed in one hour. The remaining solution acts as a reserve, the gradual absorption of which explains the more protracted improvement as compared to results obtained by other methods.

He had used the other methods at the Infants Summer Hospital, but this year he chose the intraperitoneal route for children who had lost large amounts of fluid by vomiting and diarrhea. It proved superior to all other methods because of the ease and rapidity of administration, the volume of fluid that can be given at one time, and the certainty that no fluid will be lost. The results from this treatment are remarkable; and although it has been used only in the most serious cases, the results have been most satisfactory.

The fluid carried the child over the critical days until the bowel condition began to improve. He had never before seen a child recover as quickly after so long and severe an illness. The recovery is evidence of the value of this method of treatment and of the safety with which repeated injections may be made through the abdominal wall.

Electricity

Electricity as a remedial agent in the treatment of diseases of children is employed in the following forms, in order in which they are named: Galvanic, faradic and static.

The Galvanic Current.—The effect of the galvanic or direct current on the *muscle* is to produce contraction. The contraction takes place at the moment the current is closed or opened (make or break). The galvanic current, if applied by means of two electrodes along the course of a *motor nerve*, produces a uniform contraction of the entire muscle supplied by that nerve. The reaction produced by the constant current upon the *sensory nerve* varies according as the application is made with the positive or negative electrode, the anode being sedative in its effects, the

cathode stimulating. A constant current of suitable strength—10 to 15 milliampères—passed through living tissues causes, at the point of contact of the anode, an accumulation of oxygen, chlorine and acid; coagulation and shrinking of the exposed tissue—*positive electrolysis*. On the other hand, if the cathode is brought in contact with living animal tissue, hydrogen and the alkalies are set free, and liquefaction of the parts adjacent to the electrode takes place—*negative electrolysis*.

The Faradic Current.—The faradic or induced current causes contraction of muscles and nerves and is very effective in producing muscular massage. It stimulates nerve action and nutrition, excites secretion, and arouses latent physiologic function.

The Static Current.—The static current produces vivid and persistent contraction of a large group of muscles with a minimum of pain. The second prominent characteristic of this current is its power of relieving pain. The same applies to the ultra violet rays.

The following rules should be borne in mind:

1. Always administer the weakest possible current that will cause muscular contraction.

2. Never employ electricity in the inflammatory stage of organic disease.

3. In applying electricity to muscles always endeavor to reach separately the electromotor points. In deep-seated muscles the current should be applied along the course of the nerves supplying them.

4. Each electric treatment should last no longer than twenty minutes, and no one muscle should be subjected to the currents for more than three minutes.

The indications for electricity in the treatment of diseases of children are practically the same as in adults. The discussion of the subject will, therefore, be limited to diseases in which electricity is of undoubted value.

Chronic Constipation.—The galvanic or faradic current may be used. One electrode is passed successively over different portions of the abdominal wall, and the other electrode is placed upon any other part of the body. The electric treatment should be continued for a long period.

Diphtheritic Paralysis.—In this condition, faradization of the respiratory muscles, particularly of the diaphragm, is of some service. It should be used in attacks of respiratory failure and continued while they last.

Enuresis.—The broad anode is placed over the lumbar region of the spine and the small cathode over the region of the bladder or upon the perineum, allowing quite a strong galvanic current to act for from two to four minutes. Sometimes faradization proves effective. The wire

end of the conducting cord, connected with the negative pole, should be introduced into the urethral orifice for from 1 to 2 cm. and quite a strong faradic current allowed to act for from one to two minutes.

Facial Paralysis.—This form of paralysis is greatly benefited by a weak stabile galvanic current. It should be employed four to six times a week, for from two to three minutes at a time. The anode should be placed in the auricular fossa and the cathode placed behind the ear while the different nerve branches and the muscles are slowly stroked with the cathode. In later stages faradization also is of service.

Hysteria.—The vague disconnected symptoms of hysteria call for general electric treatment, and no form of electricity so advantageously combines tonic and sedative effects as the static current. A mild current should be employed. Two or three treatments a week will generally suffice. Galvanism and faradism also are of service, especially in hysterical contractures.

Multiple Neuritis.—The application of electricity to the affected muscles is important in order to maintain their nutrition. It should be begun after the acute stage has passed, that is, at the end of from three to four weeks. A moderate faradic current may be used if the muscles respond to it; otherwise a voltaic. The electricity should be applied daily by means of large electrodes, so that the current may reach as much muscular tissue as possible. The current should be strong enough to produce visible contraction of the muscles.

Poliomyelitis.—The galvanic current gives the best results. It should not be employed earlier than the third or fourth week. A large, flat electrode, well moistened in salt water, is placed upon the spine over the affected region and the muscles were repeatedly stroked by means of a small electrode. The current should be of such strength as will produce visible contraction of the muscles, without, however, causing severe pain to distress the child.

Rheumatism.—The sequelæ of rheumatism, atrophy and contractures often call for electric treatment. The galvanic, faradic or static current may be employed. It is sometimes advantageous to use the galvanic and faradic currents at one sitting. The treatment should be repeated at least every alternate day and continued for several months. In muscular contracture the anode should be placed over the portion of the spine governing the contracted muscles and the cathode over the muscles themselves. For the relief of pain the positive pole should be applied to the most painful spot.

Tetany.—Electric treatment has been followed by improvement in a number of cases. The stabile galvanic current should be employed:

the negative pole to the spine and the positive to the irritable nerve trunks.

Torticollis.—A weak galvanic current is frequently very serviceable. The positive pole should be placed just below the occiput and the negative pole allowed to act upon the contracted muscles for from five to ten minutes.

The indications for electrolysis are identical with those in adults.

Massage

Massage is a mechanical form of treatment consisting of intelligent manipulations of the superficial parts of the body. It is intended to produce changes in the local and general nutrition, action and other functions of the body.

Indications.—Massage is indicated in hysterical, paralytic, rheumatic and traumatic contractures of joints; in fractures, to hasten absorption of callous masses; in chronic glandular enlargements; in swellings associated with rheumatism, sprains, contusion, etc.; in torticollis, to relax muscular contraction; in constipation, atonic dyspepsia and gastric dilation; in all forms of muscular atrophy or dystrophy; as a general stimulant in cases of prolonged muscular inactivity, whether from indolence, disease, feebleness (rachitis) or prolonged use of splints or braces, or other cause; in various forms of paralysis, to improve the nutrition and function of the affected muscles.

Contraindications.—Massage is contraindicated in children suffering from gonorrheal rheumatism or peliosis rheumatica; in tuberculous, typhoid or syphilitic ulcerations of the intestines; in acute peritonitis, appendicitis, gastroenteritis, gastric ulcer; in tubercular glandular enlargements.

Massage generally includes the following principal manipulations:

Effleurage or Stroking.—In making the strokes both hands are employed. The limb is grasped with one hand just above the other, in such a manner that pressure is exerted to some extent by the whole palm, but especially the ball of the thumb and the inner surface of the last two phalanges of the fingers. The strokes are delivered in the form of an ascending spiral, the two hands being moved simultaneously in opposite directions, the lower following closely upon the upper. The strokes must be made with great regularity. Light stroking has a soothing influence; heavy stroking stimulates the superficial structures, increasing the arterial, venous and lymphatic circulation.

Friction.—This manipulation is performed with the fingertips and consists of firm circular, semicircular, or to and fro movements. It is

usually combined with effleurage and is intended to promote absorption by the veins and lymphatics.

Petrissage or Kneading and Pinching.—In kneading the endeavor of the operator is to pick up the individual muscle or muscle groups between the fingers of the two hands, or in some cases between the thumb and finger of one hand, and then to roll and squeeze the muscle with a double movement. These manipulations cause circulatory, nutritive and alterative changes in the muscles, tendons and organs within reach.

Tapotement, Percussion or Tapping.—Percussion is made either with the points of the fingers brought into a line with one another or with the side of the hand and fingers. The movement should be very rapid and elastic. These manipulations are usually employed on muscular parts, such as the back of legs and gluteal regions. The effect of tapotement is similar to that obtained by petrissage. This manipulation may be enforced also by vibrations, that is, by rhythmic, tremulous movements under pressure.

Generally, all the movements are practiced at one sitting: thus, effleurage, friction, petrissage, tapotement and vibration. The treatment is concluded by effleurage. While in local affections local massage is generally sufficient to effect the desired results, it is always advantageous to supplement the local treatment by general massage. The duration of each seance varies from a few minutes to a quarter of an hour. At first the treatment should not last more than five minutes. No force should be used, and the delicate skin of the child should be spared unnecessary injury. It is, therefore, advisable to anoint the skin with boric acid vaseline, cocoanut oil or any other emollient. In young infants massage should be limited to general friction of the body. In cases of malnutrition it is a good rule to give a fat inunction daily after the morning bath.

Climatotherapy

Change of climate has from time immemorial been recognized as a therapeutic measure *par excellence*, and, fortunately, our great country abounds with vast mountain, seashore and inland resorts, which rival, if not surpass, the most celebrated spas of Europe.

In selecting a suitable health resort, we should bear in mind not only the state of health and the peculiarities of the individual patient, but also the local conditions of the particular resort, such as the drainage, water supply, prevalence of epidemic or endemic diseases, etc.

The air of mountainous regions is rarefied, dry, cool, bracing and free from organic and inorganic impurities. It improves the action

of the skin; favors deeper expansion of the lungs, and correspondingly accelerates the heart's action, improves sleep and stimulates the appetite and the powers of assimilation. Mountain air, therefore, is particularly beneficial in chronic disorders of the alimentary tract and liver; in anemia; in divers respiratory affections; in malaria; in rheumatism, and compensating heart disease.

The climate of the seashore is pure and very strong. The air is loaded with moisture, and comparatively free from dust particles, hence very beneficial to convalescents from pneumonia, pleurisy and empyema; also typhoid and surgical operations. It often acts almost specifically in acute gastroenteritis of infants.

The surf baths are invaluable in cases of nervousness, rachitis and local tuberculosis.

Dry, sheltered inland resorts are to be preferred for patients suffering from noncompensating heart disease, severe bronchitis, chronic kidney disease, and all such affections as are apt to be badly influenced by sudden variations of temperature.

It is often of advantage to spend part of the summer months at the seashore and part time in the mountains or inland resorts. Young children suffering from tuberculosis will, during the winter months, derive the greatest benefit from a sojourn in New Mexico and Arizona. Children over ten years old often do well in colder climates, such as the Adirondacks.

Select Medication in Children

In the practice of medicine, in contradistinction to surgery, every physician, I believe, passes through three well-defined psychologic experiences in the first few years of his momentous career. Overwhelmed by the enthusiasm over the infallibility of drugs as impressed upon him during his college days by the learned professor of materia medica, he enters the medical arena with boundless confidence in his power to cope with every phase of disease and anxiously awaits the opportunity to demonstrate and reap the benefit of his skill. In the early period of his career he may luckily find encouragement for his belief and anticipations, rarely, if at all, surmising that many of the cures he happened to effect were in reality only natural or accidental events, often based upon false premises, erroneous diagnoses. Lo and behold! A few unexpected failures—and his fantastic dream is cruelly shattered.

With multiplying failures the state of exultation is gradually replaced by that of depression, his hyperoptimism changes into hyperpessimism. His growing skepticism in the efficiency of medicines often

leads him to seek unalloyed self-sufficiency in one of the many fields
of surgery. Or, if he is over susceptible to the pricking of his con-
science, he even goes so far as to abandon his profession entirely,
little realizing that no profession, trade or business has attained the
millennium of righteousness and immaculate dealing with his fellow-
men. Some physicians, although continuing to practice medicine per-
manently, float in the "river of doubt," prescribe nostrums and *place-
bos,* or fall into the trap of the polished patent medicine mercenary or
promoter of some newly discovered, but invariably threadbare, mechani-
cal device or manipulation, or spiritual panacea, cult or science.

Fortunately, most of us are not so readily swayed from the straight
path of duty by such melancholy philosophy. On the contrary, we
continue conscientiously to minister to the sick and to strive to perfect
our knowledge in accord with the scientific advances of modern medi-
cine. Indeed, as with many years of hard work and careful study
our powers of intuition and judgment improve and we acquire a higher
degree of skill to select the most appropriate remedial measures to
combat disease, we soon find ourselves in the happy psychic state
of equanimity and self-reliance and content with our noble mission,
undaunted by occasional failures and untainted by triumph, fully
conscious of the limitations of medicine, yet perfectly confident in our
ability to relieve suffering, to prevent disease and to prolong life.

Those of us who believe in the therapeutic value of the drugs they
prescribe, must first of all see to it that their patients are able to
swallow and retain them. As a rule, adults manage by means of cap-
sules or condiments to render medicines, disgusting in taste, either
tasteless or at least acceptable. On the other hand, children are com-
pelled to take the medicine as given to them, and what is still worse,
the more they resist, the more they are subjected to anguish and dis-
tress, nay, even to severe corporal punishment, which not rarely
borders on serious injury.

Indeed, it is not at all rare to find little children suffering from
acute lung or heart disease in a state nigh to suffocation from the
effects of prolonged and firm compression of the nostrils, and many a
helpless child bleeds from the lips and gums and even loses a tooth or
two in the struggle with the overzealous mother who is determined
to force down its throat a teaspoonful of a miserable decoction, which
was, perhaps, intended only as a *placebo.* There is certainly no excuse for
such cruelty, much less so in the present state of pharmaceutical progress,
which enables us to select and to administer the most potent drugs in
concentrated and palatable form.

For the sake of convenience, and in order to avoid unnecessary repetition, the usual classification of drugs in accord with their therapeutic action will here be followed.

Digestants

Except in combination with other drugs digestants are rarely needed in the treatment of diseases of children. Occasionally pancreatin is indicated in starch indigestion of infants, and may be prescribed either in powder form with bicarbonate of soda or the diastatic essence, with or without a small quantity of milk of magnesia. The latter combination mixed with glycerine and fennel-seed water will be found useful in colic. The elixir digestivum compositum (N.F.) serves as a very palatable vehicle.

Tonics

The simple bitters fully deserve their striking cognomen, since they are surely very bitter and simple, insignificant, in therapeutic action. I believe that the tincture of gentian, quassia, calumba and even cinchona owe their trifling medicinal quality to the alcohol they contain. Their use in children, therefore, is hardly to be commended. Whenever a bitter tonic is desired, we should preferably resort to a minute dose of nux vomica.

Of the so-called aromatic bitters, eucalyptol is the only preparation worth mentioning. Internally it may be administered in one or two drop doses, thoroughly mixed with honey, glycerine and mucilage, in case of spasmodic asthma or laryngitis, and in the same affections it is very useful as an inhalation, especially if combined with the compound tincture of benzoin.

Quinine, which is erroneously classed among the peculiar bitters, is, of course, indispensable in the practice of medicine. Owing to its miserable taste, it is, unfortunately, not receiving as wide an application as it fully merits. We are all familiar with its specific action in malaria, but it is also a sovereign remedy in pertussis, pneumonia with delayed resolution, and in irregular or chronic grip. I am not prepared to say whether or not the brilliant effect in these cases is possibly due to some latent malarial disposition. Its miserable taste, as already stated, often precludes its administration to young children, for disguise it as one may, quinine will always taste after quinine as long as there is quinine in the mixture. Some time ago* I suggested the rectal administration of bisulphate of quinine, but, while this method works exceedingly well in hospital practice, it is not very

---

*N. Y. Med. Jour., Oct. 23, 1897.

ideal as a routine procedure. Where prompt results are desired we do best by giving it by mouth. The bisulphate is dissolved in water and rendered at least acceptable by the addition of extractum glycyrrhizæ and syrupus acaciæ. In severe cases of malarial fever characterized by excessive vomiting and pronounced nervous symptoms, we have to resort to the hypodermic. Five grains of quinine hydrochloride dissolved in 15 drops of hot water forms a suitable dose and may be injected intramuscularly under most careful aseptic precautions, two or three times daily. In pertussis quinine bisulphate may be given in one or two grain doses every two to four hours, whereas one or two large doses of quinine often suffice to hasten resolution in pneumonia or grip. Children over five years of age can often be induced to swallow chocolate coated tablets.

Of the numerous iron preparations in the pharmacopeia preference should be given to the tincture of chloride of iron, the solution of peptomanganate of iron, the syrup iodide of iron, and the dried sulphate. Iron is always useful for children and especially for infants fed exclusively on milk, which, as is known, is poor in iron, and the solution of peptomanganate of iron will be found to act exceedingly well in all simple anemias of infancy. The tincture chloride of iron is usually prescribed as a styptic and hematinic in tonsillar affections and is advantageously administered in one or two drop doses in combination with the tincture of myrrh, potassium chlorate and glycerine. This mixture adheres more or less closely to the tonsils and thus exerts its astringent effect upon them, often dispensing with gargles and local applications. The syrup iodide of iron is an ideal hematinic tonic in children, more especially in secondary anemias following or complicating acute infectious diseases, rachitis and diverse forms of glandular enlargement. In the so-called scrofular affections it often acts almost specifically, more particularly if combined with codliver oil. We have ample reason to believe also that this preparation, in addition to local attention to the nasopharynx, is frequently instrumental in reducing or even entirely removing adenoid vegetations, and its administration may be highly recommended to children who, notwithstanding the operative removal of the adenoids, continue to suffer from persistent catarrh of the respiratory tract, and show a marked disposition to repeated recurrence of the adenoids. Finally, it is worth emphasizing that owing to the destructive effect of liquid iron upon children's teeth, powdered iron with a little sugar or in tablet form should be given instead, whenever possible.

The selection of a stable and palpable phosphorous preparation is quite a problem; hence its use in children is usually limited to its

derivatives. The syrup of lime and soda hypophosphites will be found particularly beneficial in rachitis and associated affections, such as tetany and eclampsia infantum. It may advantageously be combined with syrup iodide of iron and codliver oil, which contrary to all expectations is taken by young children with considerable delight.

Mineral Acids

Insufficient attention is being paid to the medicinal properties of mineral acids. Aside from its usefulness in anacidity the dilute hydrochloric acid will be found extremely serviceable in all protracted fevers, such as typhoid and in tuberculosis. In anorexia of children dilute hydrochloric acid combined with essence of pepsin and small doses of nux vomica often works wonders. The dilute nitromuriatic acid in from 2 to 5 drops, well diluted, is indicated as a preventive of the so-called bilious attacks, characterized by recurrent vomiting, headache and catarrhal jaundice, and the syrup hydriodic acid is an invaluable remedy in all chronic bronchial affections of children, more particularly in unresolved pneumonia and asthma.

Alteratives

Arsenic, iodine and mercury form the standard remedies of this group, and if given in ample doses are invariably productive of excellent results. The use of arsenic in children is generally limited to chronic blood affections and chorea. In blood diseases arsenic should be administered in combination with iron either by mouth or hypodermically where prompt action is desired. For this purpose iron arsenate, from gr. $\frac{1}{4}$ to gr. 1, will be found particularly beneficial. From time immemorial arsenic has been lauded as a specific in chorea, and all of us have had occasion to corroborate this view. It is well to bear in mind, however, that arsenic is practically useless in the so-called rheumatic or infectious variety of chorea which calls for absolute rest in bed and salicylates, and may prove to be a very grave affection if procrastinated by arsenic treatment. In the neurotic type of chorea Fowler's solution may be pushed to its full physiologic effect, provided the urine is carefully watched for a possible renal irritation. Fowler's solution in small doses seems also to enhance the therapeutic value of the bromide in the treatment of epilepsy.

Except in syphilitic affections, the syrup iodide of iron or hydriodic acid should be given preference to potassium and sodium iodide. Moreover, it is worth noting that the iodide *per se*, *i. e.*, without mercury will never cure syphilis, be it congenital or acquired. Hence, the sooner mercury is resorted to, the better for the patient. The iodides

may be rendered more or less palatable by means of peppermint or orange flower water and simple syrup, or fluid extract of sarsaparilla and water.

Mercury is the specific in syphilis and may be administered in children by inunction, fortified by protiodide of mercury internally. Five to 10 gr. of a 50 per cent unguentum hydrargyri in lanolin, rubbed in thoroughly once a day, and from 1/16 to 1/12 gr. protiodide of mercury three times daily, will show beautiful results in a very short time. In the beginning of the treatment it may be necessary to administer a few drops of paregoric daily to allay intestinal irritation. In the newborn with congenital syphilis we may at first order 1/10 gr. calomel every three hours, and follow it up with the aforementioned remedies a few weeks later. There is very rarely any occasion in children to use more vigorous methods of treatment.

Antipyretics and Antirheumatics

Water internally and externally is the best antipyretic in children. Whenever the temperature is ephemeral in character, as for example, in indigestion, tonsillitis and the like, a cold sponge or pack answers the purpose admirably. On the other hand, when the temperature is continued and recalcitrant an effort must be made to influence the cerebral heat center, and this is best accomplished by means of warm tub baths. They tranquilize the nervous system, equalize the circulation, produce diaphoresis and reduce the temperature without shock or depression. However, in highly nervous children, antipyretic drugs, such as phenacetin, antipyrin, etc., are also indicated, and if given in moderate doses at long intervals are perfectly harmless. They may be made fairly palatable in syrupus acaciae and orange flower water. It may here be emphasized that a moderate dose of an antipyretic will often promptly control an attack of convulsions in children, at any rate long enough until its cause has been determined and appropriate remedies employed for its permanent removal. It is well to remember also that small repeated doses of antipyretics, more particularly pyramidon, will frequently subdue grotesque choreic movements where the usual treatment utterly fails. The specific of salicylates in rheumatic affections is too well known to require reiteration on my part. It may be noted, however, that salicylates are tolerated by children in larger quantities than by adults, and if administered with a little caffeine sodium benzoate or strophanthus are perfectly free from depressing after effects. On many occasions I have had the opportunity to convince myself, as well as others, of the distinct abortive powers of sodium or ammonium salicylate in acute poliomyelitis. The

salicylates are also invaluable in all acute infectious diseases; and whenever one is in doubt as to what medicine to prescribe, one will almost invariably strike it right by selecting this remedy. It, furthermore, has the good quality of being palatable.

Hypnotics, Anodynes and Antispasmodics

The selection of pleasant hypnotics and anodynes is rather difficult, and perhaps fortunately so, since their effect upon the delicate infantile organism at best is more or less deleterious. Very recently I was consulted to see a newborn supposedly suffering from atelectasis pulmonum. The baby was in profound stupor, its pupils markedly contracted, its breathing from ten to twelve per minute, and pulse from forty to fifty per minute, barely perceptible. It refused to nurse and swallowed with difficulty. The family physician informed me that the baby had been sneezing and coughing, and to relieve the anxiety of the parents he had prescribed a cough mixture containing 5 minims of paregoric in each dose, which was administered every two hours. Obviously we were dealing with a case of opium poisoning. By promptly directing the treatment against it the infant recovered very rapidly. The safe dose of paregoric for children is one drop for every year of the child's age, and one tenth of this quantity when the tincture of opium is prescribed. In gastrointestinal affections where an opiate is indicated, preference should be given to Dover's powder (one-tenth of a grain for every year of the child's age), because of the beneficial effect of the ipecac it contains; and whenever vomiting precludes its administration, we will often find an opium suppository to act admirably. Where very prompt action is desired, as for example, in cholera infantum with profuse vomiting and purging, we may advantageously resort to a hypodermic injection of morphine (gr. 1/60) and atropine (gr. 1/600). Morphine and atropine hypodermically are occasionally indicated also in other acute diseases, e. g., uremic convulsions. In respiratory affections codeine and its similar morphine derivatives are the drugs par excellence. Whenever a hypnotic is indicated, codeine added to bromide will act by far better than the newer coal tar byproducts. The use of spasmodics in children is somewhat limited. In olden times belladonna was looked upon as the *sine qua non* in pertussis. It was pushed to its full physiologic effect—until the child was practically blinded. We know better today. Belladonna will be found useful, however, in ordinary catarrhs of the respiratory tract with profuse mucous discharge and in rhinitis of infants when the nasal discharge interferes with nursing. In combination with codeine, in the form of suppositories, belladonna is of particular value in irritable

bladder, strangury and tenesmus, and also acts nicely as a palliative in catarrhal appendicitis, when medication by mouth is contraindicated. We may choose hyoscyamus instead of belladonna; in fact, there is no better antispasmodic and anodyne in dysuria accompanying cystitis than hyoscyamine sulphate. The dose is gr.1/800 for every year of the child's age, dissolved in syrupus altheæ and water.

Stimulants

Practically all stimulants are unpleasant in taste and require skillful compounding to render them palatable. Glycerine and the elixir digestivum compositum serve best in this direction. Strychnine and strophanthus are indicated in almost all infectious diseases of children, and should be administered early rather than late. In the early stage of the disease the dosage, of course, should be small. Gr. 1/300 of strychnine and m. 1 of tincture of strophanthus for every year of the child's age will ordinarily serve the purpose admirably. I believe it is a mistake to leave stimulation in pneumonia to the very end, and I have made it a rule to give strychnine and ammonium carbonate or liquor ammonii anisatus the first three days of the disease, strychnine and strophanthus the following two or three days, and digitalis and alcoholic stimulants in the last days when the cumulative effect of the pneumonia toxin is most apt to undermine the cardiac muscles. In urgent cases we are often called upon to add caffeine sodium benzoate and even adrenalin. It is always a good plan to provide the nurse with an ample supply of quick stimulants for an eventual emergency during the crisis, and I am able to assure the reader that at least in one case this intensive preparedness has worked wonders. About two years ago I was invited to see a nine year old boy suffering from grippal, so-called wandering pneumonia of two weeks' standing and complicated by double otitis media. As the patient was extremely weak and showed distinct signs of myocardial involvement, I suggested to the family physician to supply the nurse with three additional hypodermic syringes, one containing 10 drops of adrenalin, the second 5 gr. of caffeine sodium benzoate, and the third 1/30 gr. of strychnine, so as to be on guard against sudden heart failure. The next day another consultant was called and the day following a third one. This excellent clinician, in his laudable effort to ascertain the cause for the delayed resolution, directed the nurse to sit the boy up in bed in order carefully to examine the posterior portions of the thorax. While doing so the patient suddenly flapped backward, his jaws dropped, his pupils dilated and his heart stopped beating, in short he seemed as dead as a doornail. The physicians became terrified, mortified and left the sick-room to con-

vey to the child's parents the dreadful message of the unfortunate event. In the meantime the nurse recovered sufficient sense to proceed with the simultaneous injection of the three aforementioned reserve stimulants in the boy's arm. To her great delight she soon noted a slight twitch of his mouth and heard a faint fluttering of his heart, and in another few minutes the boy was himself again. Resolution set in the following day. For a number of years past the profession has placed a great deal of reliance on sterile camphor oil as a powerful stimulant, more especially in pneumonia. I have used it extensively and am still resorting to it occasionally, but must frankly confess never to have been convinced of its utility and have often felt that the sorely tried patient ought to be spared the pain and discomfort almost invariably associated with its hypodermic administration. Indeed, I fear, that the needle has been recently grossly abused, be it in the subcutaneous injection of drugs or of an unlimited number of inert vaccines and serums, which in the majority of instances serve only to fill the coffers of mercenaries. It is my hope that these remarks will not be misunderstood. While firmly believing in the life saving properties of antidiphtheritic and antimeningococcic serums and the like, I cannot help but feel that the profession is entirely too credulous to the exaggerated threadbare claims of the vaccine manufacturers. In treating heart disease, it is well to remember that while digitalis is the indispensable remedy in chronic cases with "ruptured compensation," it is more or less harmful in acute heart disease, with compensation intact, when an ice bag to the precordium and small doses of codeine with or without sodium salicylate are indicated.

Heart Sedatives

If ever there be any need for heart sedatives in children, we could readily get along with a minute dose of morphine or its derivatives or possibly some coal tar product, such as pyramidon. Aconite, the standby of the homeopath, similar to digitalis is a dangerous drug in the hands of the inexperienced. The real indication for aconite is supposed to be sthenic fever, and there are not many children too vigorous while ill. However, in homeopathic doses and well diluted, it probably can do no harm.

Emetics

No great effort need be made to disguise the taste of emetics. The wine of ipecacuanha, requiring but small doses, should be preferred to the syrup, and whenever emesis is very urgent a hypodermic in-

jection of from gr. 1/12 to gr. 1/16 of apomorphine will prove most satisfactory. It is to be regretted that emetics are dropping into disuse, for many a case of acute indigestion in children could promptly be arrested by swift emesis. It may be worth mentioning also that moderate doses of ipecac are invaluable in whooping cough with prolonged suffocating paroxysms, thus by emesis imitating nature in aborting the attack.

Expectorants

The selection of suitable expectorants requires good judgment. It is useless, in fact harmful, for instance to prescribe stimulating expectorants such as ammonium chloride or carbonate in cases of persistent coughing arising from nasopharyngeal or laryngeal inflammation. We should rather be inclined to allay the source of irritation by local measures and administer a sedative to relieve the cough. With this object in view excellent results are usually obtained from daily instillation of from 2 to 5 per cent of argyrol, solargentum, or silvol in the nose, and the internal administration of creosote carbonate and codeine, well mixed with glycerine mucilage and water. On the other hand, when dealing with a harassing cough in acute bronchitis or pneumonia in which the expectoration is very adhesive and cohesive, scanty in amount and hard to raise, a stimulating expectorant with or without wine of ipecacuanha or compound syrup of squills, in the majority of instances will prove beneficial to the patient by assisting nature to rid the lungs of effete material and save the patient's energy in the terrible battle ahead of him. In chronic bronchitis and unresolved pneumonia satisfactory results are frequently achieved from ammonium iodide, gr. ½ to 1 for every year of the child's age, every three or four hours, or from the syrup of hydriodic acid or syrup iodide of iron. The iodides are very useful also in the exhausting cough accompanying noncompensating heart disease, and may advantageously be combined with digitalis and an occasional dose of some morphine derivative.

Diuretics and Diaphoretics

Water is the most palatable and, in large quantities, the most efficient diuretic in bladder affections. If given hot and sweetened with sugar it is also an active diaphoretic and should be given in preference to offensive diaphoretic mixtures, whenever possible. When drugs are needed, potassium acetate and citrate generally serve well both as diuretics and diaphoretics. Potassium citrate in combination with hexamethylenamin acts specifically in pyelocystitis. Considerable

caution, however, is commended in the continued administration of the latter, owing to its tendency to produce hematuria. Another excellent preparation is sodium benzoate which forms an ideal diuretic, diaphoretic and expectorant in the group of symptoms frequently encountered in acute influenza.* Diuretics are practically useless, nay, often harmful, in acute nephritis while the urinary tubules are obstructed by the inflammatory changes. In such cases we do much better with active diaphoresis by means of packs and hot baths and intestinal flushing, to rid the system of effete material without overburdening the renal function. On the other hand, in subacute and chronic nephritis, diuretics must be resorted to whenever the excretion of urine is diminished and a tendency towards dropsy becomes manifest. In such cases the liquor ferri and ammonii acetatis seems to act exceedingly well. If, however, the dropsy is extensive and of cardiac origin, we always have to fall back on digitalis and strophanthus with or without diuretin or theocin sodium. Another indication for free diuresis is pleurisy with effusion, especially if the medication is coupled with complete abstention from all fluids in the diet. Under these conditions nature seems to absorb the fluid from the pleural sac to replenish the needs of the human economy.

Laxatives and Purgatives

We are all too familiar with the action of castor oil, calomel and phenolphthalein to require any detailed discussion. Attention may here be directed to the fact that there is no particular reason for giving calomel in divided doses and bothering the child unnecessarily. Any baby can stand a grain of calomel without much ado. Effervescent citrate of magnesia is contraindicated where there is a tendency to vomit, the milk of magnesia being by far preferable. Purgatives and hydragogues are indispensable in acute nephritis and dropsical effusions when the kidneys are unable to perform their function; unfortunately, however, all these preparations are very disgusting in taste and require large quantities to produce the desired effect. Rochelle and Epsom salts may be rendered fairly palatable by the addition to a saturated solution of about a third of its quantity of glycerine and a few drops of aromatic spirits of ammonia. The mixture may then be administered in teaspoonful doses at frequent intervals. The infusum sennæ compositum may prove useful in some cases, but we must see to it that it is freshly prepared. In treating chronic constipation of children our efforts in the direction of regulation of the diet and induction of regular habits will only too often fail,

*Influenza in Children, N. Y. Med. Jour., June 30, 1900.

and we are frequently called upon to advise a suitable laxative. Malt extract with olive oil, or cascara, or mineral oil will answer the purpose in the majority of cases.

Intestinal Astringents

Most of us still recall the sad time when during the summer months every pharmacist was stocked up with a large exhibition of summer-complaint-mixtures to meet the great demands of the season. Fortunately, with our advanced knowledge of the causes of the summer diarrheas of infants and the methods of prophylaxis, there is no longer any excessive demand for such preparations. However, we have not as yet reached the millennium in infant feeding and hence are still called upon to prescribe the time worn, yet efficient, bismuth and chalk mixtures. Ordinarily I prefer bismuth subcarbonate to the subnitrate, and order to give the patient from 10 to 20 grains after each evacuation. In this manner the dosage is controlled in accord with the severity of the diarrhea. As already stated, in colitis and cognate affections opium and ipecacuanha will be found to act most satisfactorily. Dover's powder may be made palatable by the addition of pulvis aromaticus. The different tannin preparations are worthy of trial only in chronic enteric affections, but here greater benefit will be derived from local treatment, more particularly daily intestinal irrigations with ½ to 1 per cent of nitrate of silver, in addition, of course, to an appropriate diet. Sodium bicarbonate is, of course, the specific in diarrhea associated with acidosis or fat and sugar indigestion, and should be given in large doses by mouth as well as per rectum.

Gastric Sedatives

Last in line but foremost in importance are the gastric sedatives, since with a highly irritated stomach, when all food or medication is promptly ejected, even a very mild disease *per se* is most apt gravely to undermine the baby's power of resistance; hence, the importance of first of all settling the stomach. In the majority of instances this is readily accomplished by one large dose of sodium bicarbonate, let us say, from 30 to 60 grains in water and followed up by smaller quantities of bicarbonate of soda and subcarbonate of bismuth with or without calomel. Good results are often obtained also from the different medicated waters, such as lime, peppermint and bitter almond water in cracked ice or 1/20 of a drop of tincture of iodine well diluted. In recurrent vomiting lavage is indispensable, and in some cases we may even have to resort to a hypodermic injection of mor-

phine. Of course, in all cases of severe vomiting careful attention must be given to its etiology, more particularly involvement of the appendix or brain, or acidosis.

In administering medicines to children, it is often helpful to divide the full dose in several small doses, if need be, giving it drop by drop until the whole teaspoonful has been taken. In this manner even a most irritable stomach will often retain the medication, whereas it would otherwise reject it.

In conclusion let me suggest the following general rules to facilitate the selection and administration of drugs to children:

1. Never prescribe any medicine unless you are convinced of its necessity; if only a *placebo* is required, prescribe a palatable adjuvant.

2. Never prescribe a medicinal preparation requiring a large quantity, when a small one of the same or an equally as useful a drug will do the work just as efficiently. Thank Heaven, the time is past when the greatness of the physician stood in direct ratio to the quantity of the concoction he ordered.

3. Never prescribe a painful therapeutic procedure or a nauseous mixture when the patient will do equally as well—and surely much better—without the unnecessary pain and annoyance.

Organotherapy

Organotherapeutics, though still in the experimental stage, is rapidly assuming an enviable position in the field of specific medication. This is true especially of the thyroids, and less so of the suprarenals, pituitary and thymus glands.

Their *modus operandi* upon the human economy—whether by regulation of metabolism, or neutralization of specific poisons—is still shrouded in mystery. It is definitely established, however, that they are all of fundamental importance to the health and growth of the human organism.

From a therapeutic point of view the thyroid gland only has thus far met all expectations. It acts specifically in cretinism and myxedema, and is very serviceable also in obesity and pachydermatoses. The gland may be administered fresh (in soup) or dry. The dry preparations are usually given in from $\frac{1}{2}$ to 3 grain doses twice daily, until the desired results have been obtained, and in smaller quantities thereafter. Engrafting of the sheep's thyroid in the human body has met with some success. The parathyroids are generally employed (gr. $\frac{1}{10}$ to $\frac{1}{4}$) as adjuvant or substitute of the thyroid.

The suprarenal solutions are used principally locally as hemostatic and astringent, e. g., epistaxis, rhinorrhea of divers origin. Internally,

usually hypodermically (5 min. of a 1:1000 solution) in heart failure and to abort a severe attack of asthma.

The pituitary gland is (gr. $\frac{1}{4}$) highly recommended in infantilism, in hay fever and asthma (topically as well as internally), in diabetes insipidus, enuresis, and tympanites (hypodermically).

The therapeutic application for the thymus gland is thus far limited to pronounced anemias and marasmus. The results are encouraging.

More recently the pineal gland has been found of service in diverse forms of mental deficiencies, especially Mongolian idiocy (p. 718).

Vitamines

Vitamines are vital food substances belonging to a group of organic bases of unknown composition which seem to be essential to metabolism and the maintenance of good health and normal growth and development of the body. They are believed to be closely allied to hormones (pancreatic secretin) and possibly also to enzymes. Some vitamines are soluble in water and others in fat, the latter being probably closely related to lipoids. It is claimed that lack of vitamines forms the underlying cause of rachitis, scorbutus, beriberi and pellagra, but as yet there is no positive clinical evidence to confirm this view, although laboratory experiments tend to favor it. Autolyzed yeast (see p. 517) is being used as a vitamine in the aforementioned affections.

CHAPTER II

EXAMINATION OF THE PATIENT
AND
SEMEIOLOGY OF DISEASE

A successful physical examination of a child, especially of an infant, calls for a great deal of tact, patience, and careful scrutiny. The physician will do well to train his eyes at a glance to observe and to interpret the aspects of disease. As will be noted later, in a large number of diseases, the attitude, the facial hue and expression, the size and shape of the child or of some parts of the body and finally the aspect of the skin, teeth, etc., are often pathognomonic. This general survey is preferably made while the patient is still undisturbed, utilizing the same time for gathering the most essential points of information pertaining to the family, past and personal history of the patient.

Family History.—Longevity of the parents, brothers and sisters; the diseases they suffered from, especially as to tuberculosis, syphilis (miscarriages in the mother often more decisive in the diagnosis than the Wassermann test!), rheumatism, heart, kidney or liver disease, alcoholism, epilepsy, insanity, etc.

Past History.—Degree of maturity at birth, and mode of delivery (instrumental or otherwise); condition soon after birth, particularly as to signs of traumatism, convulsions, asphyxia, deformity, hemorrhages, skin eruptions, nasal catarrh ("snuffles"); the diseases the patient suffered from at a later period, e. g., gastrointestinal, exanthematous, pulmonary; otitis, rheumatism, bone affections, etc. Mode of feeding (breast or bottle), gain or loss of weight; time of eruption of temporary or permanent teeth; the time when the patient began to sit up, stand, creep and walk. Peculiarities of temper, etc.

Present History.—Age of patient.

Mode of onset of the disease (gradual or sudden).

Fever (continuous, remittent or irregular).

Convulsions[1] (apparent cause; time of occurrence; duration).

Vomiting[2] (during, after, or between meals; appearance of vomit).

Skin eruption[3] (location, duration; desquamation).

Diarrhea[4] (duration; frequency and appearance of the stools).

---

[1]See page 669. [3]See page 398.
[2]See page 156. [4]See page 157.

Constipation[5] (acute or habitual; appearance of the stools),

Pain[6] (situation, duration; degree of severity).

Cough[7] (duration; paroxysmal or croupy; appearance of sputum).

Dyspnea[8] (worse after fatigue or at night; sudden).

Cyanosis[9] (duration; mode of onset—with convulsions).

Urinary disturbance[10] (enuresis, dysuria, suppression; appearance of urine).

Disturbance of Sleep (pavor nocturnus; snoring; twitching; crying from "starting pain").

Behavior and Mental Capacity[11] (recent changes, if any).

Condition of Special Senses[12] (defective vision, hearing, etc).

The history taking completed, we next turn to the physical examination of the patient. This should be systematic, preferably with the child entirely undressed, and if deemed necessary, should include inspection, palpation, auscultation, percussion, mensuration and weighing.

We usually begin with the examination of the head, noting its size and shape, the condition of the bones of the skull, its fontanelles and sutures, its attitude; facial expression and hue; condition of the nose, eyes, ears, mouth, lips, tongue, teeth and pharynx.

THE HEAD

The head is rarely normal in *shape* immediately after birth. The scalp is swollen, the bones are often displaced, and here and there are bruises and ecchymoses, the results of a long and painful journey. Within about a week, the swelling subsides, the bones adjust themselves, the head becomes round or oval and smooth except for the markings of the fontanelles and sutures.

The *cranial circumference* (fronto-occipital diameter) soon after birth measures about 13 inches. The skull enlarges rapidly up to six months old—17 inches; then more slowly about 1 inch every year up to five years—21 inches; it then remains stationary in growth up to adult life, when it measures from 22 to 23 inches.

The posterior *fontanelle* closes by the end of the second month, the anterior when the infant is about eighteen months old, at the latest.

A healthy baby is able to hold up the head when about four months old.

The **skull** is—

1. Asymmetrical, with depressions and protrusions, in caput succe-

---

daneum; meningo- and encephalocele; syphilis; neoplasms; abscesses; oxycephalia ("sugar-loaf" head), etc.

2. Large, in hydrocephalus; hypertrophy of the brain; rachitis.

3. Small, in microcephalus; porencephalia.

The **fontanelles** are—

1. Closed late, in hydrocephalus; rachitis; cretinism; idiocy; osteogenesis imperfecta.

2. Closed prematurely, in microcephalus; atrophy of the brain.

Fig. 6.—Hydrocephalus.

3. Distended, in active and passive congestions of the brain, e. g., divers forms of meningitis; meningismus; hydrocephaloid, intracranial tumors; cerebral hyperemia.

4. Sunken, in wasting diseases; after great loss of body fluids; after lumbar puncture.

The **cranial** bones are—

1. Soft and thin, in chronic hydrocephalus; craniotabes.

2. Hard and thick, in syphilis; exostosis.

The **sutures** are—

1. Widely separated, in hydrocephalus; intracranial tumors.

2. Prematurely closed, in microcephalus.

Attitude of the head—

1. Retracted, shaky, in general debility; macrocephalus; hydrocephalus; amaurotic family idiocy.

2. Spasmodically retracted (opisthotonos), in meningitis; meningismus; encephalitis; apical pneumonia.

3. Turned laterally, in torticollis; hematoma of the sternocleidomastoid muscle; retropharyngeal abscess; cervical spondylitis; cervical adenitis; mastoiditis.

4. Moving irregularly, in hyperpyrexia; spasmus nutans; chorea; habit spasm. In eruptions of the scalp attended by severe itching.
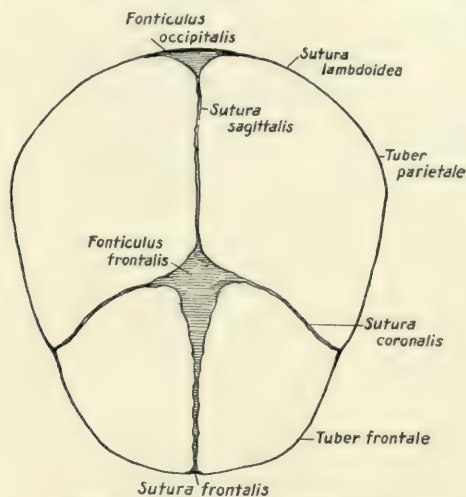
Fig. 7.—Fontanels.   (Leo-Wolf.)

The Face

Facies dolorosa—

1. Face of continuous pain (eyes open, face wrinkled, mouth half closed and drawn to one side; moaning and whining) in divers acute inflammatory diseases, *e. g.*, pneumonia, pleurisy, rheumatism, appendicitis.

2. Intermittent pain (face distorted, red, perspiring; loud crying, tossing, kicking), in colic, dysuria, etc.; vertebral caries (''starting pain'').

Facies luctuosa—

Face of sorrow (forehead and face wrinkled, face pale, emaciated, indifferent, apathetic, eyes half closed), in chronic wasting diseases, especially tuberculosis, and last stage of heart disease.

Facies anxiosa—

Face of anxiety (eyes glistening, congested, red or livid, and per-spiring; alæ nasi active), in orthopnea from various causes, *e. g.*, laryngeal stenosis, extensive pneumonia, pulmonary edema; in hysteria.

Facies hippocratica—

Face of grave abdominal distress, or extreme exhaustion (face pale, contracted, corneæ dull, eyeballs and temples deeply sunken, nose pinched, lips dry, cyanotic, and covered with sordes), in moribund state, collapse, cholera nostras, peritonitis, etc.

Facies meningitidis—

Face of internal convulsions (staring look into distance, glassy cor-neæ, rapidly changing complexion of the face), in meningitis; severe eclampsia.

Facies senilis—

Face of extreme old age (shriveled fascial muscles and skin, pointed nose, lusterless eyes), in marasmus; syphilis; chronic hydrocephalus.

Facies idiotes—

Face of the mentally defective (senile features, open mouth, pro-truding tongue), in all forms of idiocy and imbecility; less marked in adenoids.

Facies sardonica*—

Face of facial muscular spasm (peculiar "grin," proboscis-form mouth, sometimes foamy), in tetanus and similar prolonged convul-sive conditions.

Facial hue**—

1. *Livid,* in congenital and acquired heart disease; in pronounced res-piratory difficulty, *e. g.*, laryngeal stenosis, pulmonary edema, asthma, etc.; in cerebral hyperemia; sinus thrombosis; in "holding the breath."

2. *Pale,* in anemia; in acute and chronic wasting diseases; sudden pallor, in collapse, *e. g.*, from exhausting hemorrhage.

3. *Waxy,* in chronic malaria; suppurative processes; chronic nephritis; malignant disease.

4. *Yellow,* in icterus neonatorum or catarrhalis, congenital oblitera-tion of the bile duct; in Buhl's or Winkel's disease, in liver affections, especially due to syphilis.

5. *Purplish,* in phthisis pulmonalis ("hectic flush"), hyperpyrexia; pneumonia; compensating heart disease.

6. *Greenish,* in chlorosis.

---

*See also "Facial Paralysis," "Facial Hemiatrophy," "Pertussis," "Nephritis," "Trichiniasis," p. 426, "Hemiplegia."
**See also "Exanthemata" and "Skin Diseases."

7. *Copper-color* (*e. g.*, on forehead), in syphilis.

8. *Bronze color*, in Addison's disease.

The Eyes

The **eyelids** are—

1. Edematous, without local inflammation, in anemias; heart and kidney diseases; pertussis; trichiniasis.

2. Crusty, red and swollen, in acute and chronic inflammation of the eyelids; in pediculosis of the eyelashes; in congenital syphilis (in conjunction with rhagades at the canthi, and purulent nasal discharge), in scrofulosis (with keratitis, excoriation of the upper lid, and adenitis); red and watery, in nasal catarrh, hay fever, and measles.

3. Retracted, inability to lower upper lid, from loss of power in the palpebral muscles, in facial paralysis.

4. Drooping (ptosis) of upper lid, from inability to raise it, in congenital defects of the palpebral levators or their nerve supply, in local trauma; in oculomotor paralysis; ophthalmoplegia (unilateral); encephalitis lethargica.

5. Spasmodically contracting, in local inflammatory processes of the lids; in photophobia; in spasmodic affections, such as chorea and tic.

The **eyeballs** are—

1. Congested, in inflammatory processes of the eye, *e. g.*, keratitis; in meningitis; asphyxia.

2. Protruding, in exophthalmic goiter; in neoplasms (gumma); in chloroma (frog-like appearance).

3. Immobile, partially or completely, in ophthalmoplegia (unilateral).

4. Turned laterally (strabismus); in errors of refraction; in paralysis of the abducens (convergent strabismus); in paralysis of the oculomotor (divergent strabismus—with ptosis, mydriasis, and diplopia).

5. Oscillating (nystagmus), in hereditary ataxia; lesions of the corpora quadrigemina; multiple sclerosis; meningitis; sinus thrombosis; hydrocephalus.

The **pupils** are—

1. Contracted, unilaterally, in paralysis of cervical sympathetic, *e. g.*, migraine, cervical rib (may also be bilateral), in pressure by central tumor. Bilaterally, in affections of the cervical cord, both sides; early stage of meningitis; from the effects of opium and its derivatives, chloral, pilocarpine, physostigmine, etc.

2. Dilated, unilaterally, in irritation of the cervical sympathetic, *e. g.*, migraine; in oculomotor paralysis. Bilaterally, in marked dyspnea; collapse; from the effects of atropine, belladonna, hyoscyamus, cocaine, etc.

3. Unequal, in unilateral contraction or dilatation, as aforementioned; in unilateral pontine lesion, and in apoplexy.

4. Immobile, in adhesions of the iris to the lens; in eclampsia; in lesions of the corpora quadrigemina; in tabes dorsalis (immobility to light, but responding to accommodation—Argyll Robertson pupil).

Vision is—

1. Diminished, in errors of refraction; miosis; mydriasis; hysteria; acute eye affections, *e. g.*, iritis, retinitis, etc.; in corneal opacities, cataract, etc.; congenital eye defects, *e. g.*, albinism, cataract, irideremia; in toxic amblyopia, *e. g.*, overdoses of quinine, tobacco; congenital amblyopia (usually unilateral); optic neuritis.

2. Lost, temporarily or permanently, in uremic, diabetic, or other forms of toxemia; in severe convulsions of central origin; congenital
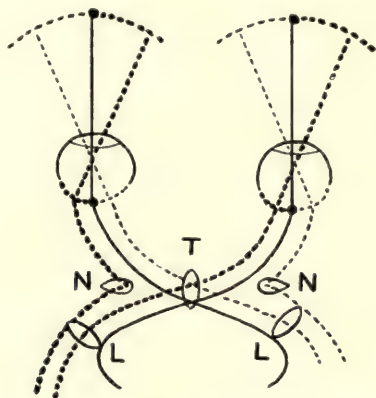
Fig. 8.—Diagram of the visual tract. *N.* Lesions producing nasal hemianopia. *L.* Lesions producing lateral hemianopia. *T.* Lesions producing temporal hemianopia.

complete cataract; amaurotic family idiocy (gradual onset); in embolism of the central retinal artery (unilateral); local injuries; optic atrophy.

3. Double (diplopia), in peripheral palsies of the eye muscles, *e. g.*, after diphtheria, influenza, herpes zoster ophthalmicus (unilateral); in strabismus. In orbital palsies, through outside pressure, *e. g.*, neoplasms. In central palsies (affecting the eye on the opposite side). In nuclear palsies, *e. g.*, of the abducens (involving the eye on the same side).

4. Half, *i. e.*, blindness of one-half of the visual field (hemianopsia); lateral or homonymous, in lesions of the optic tract between chiasm and cortex; temporal, in disease of the optic chiasm affecting the anterior or posterior angles; nasal, in disease of the chiasm affecting the outer angles (Fig. 8).

The Ears

Abnormalities of the ears and adjacent structures—

1. Asymmetry of the ears, in congenital, mentally defectives.

2. Tumefactions, at and about the ear, in the external meatus, in furuncles, abscesses, and local traumatism. In front of the ear, in epidemic parotitis (often bilateral, though not simultaneously); in secondary parotitis (complicating diseases of the mouth, local infection in the vicinity; acute infectious diseases, *e. g.*, typhoid; in new growths. Behind and downward, pushing the auricle forward, in mastoiditis; in perforating abscesses of the external auditory canal; in preauricular lymphadenitis; and much less marked in glandular fever.

Hearing is—

1. Diminished, at a distance, but not by bone conduction, in external and middle ear disease, in occlusion of the auditory canal by foreign bodies, *e. g.*, cerumen, furuncles; or outside tumors, *e. g.*, parotitis; in nasopharyngeal disease, *e. g.*, adenoids.

2. Lost, temporarily or permanently, both at a distance and by bone conduction, in congenital defects of the auditory apparatus; in compression (by intracranial tumors) or atrophy of the auditory nerve; in disease of the pons or cerebellum which has spread to the fourth ventricle; in amaurotic family idiocy.

3. Disturbed by noises (tinnitus aurium), in foreign bodies in the auditory canal, *e. g.*, cerumen, mycosis, myringitis; in catarrh of the Eustachian tube; in otitis media; neuroses; epilepsy, and mental affections.

The Nose

Abnormalities of the nose in structure and function—

1. Saddle-shaped, sunken, in hereditary syphilis; in traumatism,

2. Compressed and pointed, in nasal obstruction, chiefly adenoids.

3. Pinched and pale, in collapse; sudden fright; phthisis pulmonum.

4. Purplish in color, in circulatory and respiratory difficulties, *e. g.*, pneumonia, heart disease.

5. Hyperactivity of the alæ nasi, in grave dyspnea.

6. Nasal voice or cry, in nasal obstruction, *e. g.*, adenoids, rhinitis, retropharyngeal abscess; in diphtheritic paralysis; in ulceration of the nasal bones, especially in syphilis.

Nasal discharge—

1. Serous, transparent, later mucous, in acute simple rhinitis ("cold"); measles; hay fever.

2. Serosanguinolent, later purulent, in diphtheritic, scarlatinal, and

syphilitic rhinitis; in the presence of foreign bodies in the nose; in scrofulosis.

3. Mucopurulent or purulent, in severe acute rhinitis; in putrid infection; in sinusitis.

4. Hemorrhagic (epistaxis) in nasal trauma; inflammation of the nasal mucosa; nasal polypus; adenoids; hemophilia; vicarious menstruation; passive congestion of the brain; increased vascular tension, e. g., hyperpyrexia (especially if sudden, as it is apt to be at the onset of exanthematous diseases), heart and lung diseases, pertussis, influenza; in diseases of the blood, e. g., sepsis; leukemia, etc.

The Lips

The **lips** are—

1. Excoriated (upper lip) from acrid nasal discharge, in acute and chronic affections of the nose, e. g., rhinitis, adenoids; in scrofulosis; syphilis.

2. Covered by herpes, a vesicular eruption (usually the upper lip at the angle of the mouth) in ordinary ''colds''; in pneumonia; in meningitis cerebrospinalis.

3. Cracked and scarified, especially at the angles of the mouth, in syphilis hereditaria; but also in burns (usually unilaterally).

4. Covered by sordes, in septic infections; in typhoid fever.

5. Rosy in color, in good health.

6. Deep red, in compensating heart disease.

7. Purple, in marked dyspnea, from respiratory and circulatory disturbances.

8. Pale, in divers forms of anemia.

9. Livid, in heart failure.

10. Dirty, soot-like, in sepsis; typhoid fever; ulcerative stomatitis.

The Oral Cavity

The **mouth** is—

1. Drawn to one side, droops, in facial paralysis, especially when the facial muscles are brought into action; in progressive facial hemiatrophy; in hemiplegia.

2. Drawn outward and downward, with the lips pointed forward, proboscis-like, in trismus neonatorum, tetanus and tetany.

3. Broad and grinning, in cretinism; idiocy.

4. Large from birth, in macrostomia; small and contracted, in microstomia; in congenital syphilis; from the effect of burns.

5. Open habitually (''mouth-breathing'') in nasal obstruction; adenoids; idiocy; retropharyngeal abscess.

6. Twitching, spasmodically, in chorea; habit spasm.

Fetor ex ore—

1. Stale insipid, in catarrh of the nasopharynx; dental caries; in febrile diseases; chronic dyspepsia.

2. Putrefactive, at short range, in divers forms of simple stomatitis; acute indigestion. At a distance, in noma; malignant diphtheria or scarlatinal angina.

3. Sulphuretted hydrogen odor, in fetid bronchitis; pulmonary gangrene.

4. Acetone odor, in diabetes; cyclic vomiting; acidosis.

5. Ammoniacal odor, in uremia.

6. Chloroform, ether, alcohol, etc., odors, from the effects of these drugs.

In irritable children it is preferable to postpone the examination of the mouth-cavity until the other portions of the body have been thoroughly examined, since the undue excitement usually created by the inspection and palpation of the mouth and throat of the patient greatly interferes with the study of the other physical phenomena. Through daily practice, the physician soon learns, almost at a glance, to distinguish the abnormal from the normal; until he has acquired this skill, however, he should examine the contents of the oral cavity slowly and systematically.

The gums, teeth, floor and roof of the mouth; the tongue, buccal mucous membrane, the uvula, fauces, tonsils and posterior pharynx—all should receive careful attention.

The **gums** are—

1. Whitish, thin, and hard, normally in early infancy.

2. Reddened, slightly swollen and painful to touch, before eruption of teeth.

3. Spongy, swollen, and prone to bleed, in divers forms of stomatitis; in scurvy; purpura; in other grave constitutional diseases, such as leukemia.

4. Purulent, receding, from the teeth, in pyorrhea alveolaris; alveolar abscess.

5. Bleeding, without inflammatory symptoms, in hemophilia.

6. Colored blue, forming a blue line along the margin of the gum, in lead poisoning.

The **temporary teeth** are twenty in number, and under normal conditions generally appear in groups, at variable intervals, as follows:

1. Two lower central incisors at the age of from six to eight months.

2. Four upper incisors (2 central, 2 lateral) from eight to ten months.

3. Two lower lateral incisors from eleven to twelve months.

4. Four anterior molars (2 upper, 2 lower) from fourteen to sixteen months.

5. Four canines (2 upper, 2 lower) from eighteen to twenty months.

6. Four posterior molars (2 upper, 2 lower) from twenty-two to thirty months.

Abnormal teething—

1. Dentitio tarda, *i. e.*, considerable retardation (absence of a tooth at the age of a year or later), in rickets; general debility; congenital syphilis; cretinism; idiocy, etc.
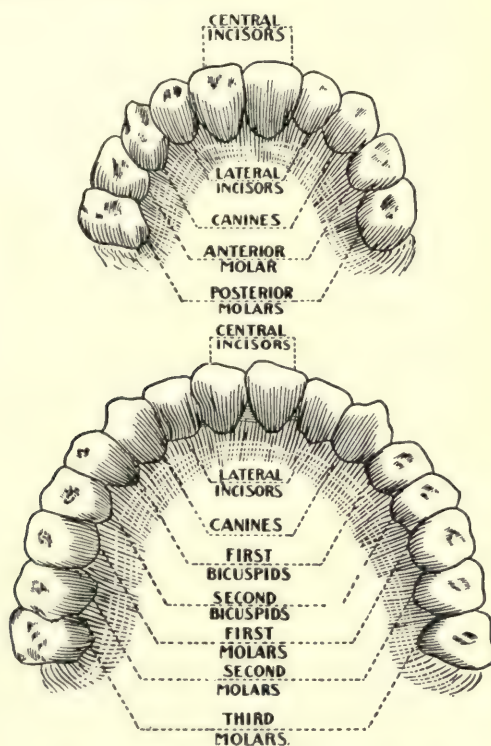
Fig. 9.—Temporary and permanent teeth.

2. Dentitio precox is of no special significance. Occasionally occurs in congenital syphilis (a tooth may appear soon after birth); in hydrocephalus.

3. Irregular implantation, incurvation, striation and premature erosion, the same as in "dentitio tarda" (*q. v.*).

The **permanent teeth** appear normally in the following order:

1. Four first molars (2 upper, 2 lower) at about six years.

2. Four central incisors (2 upper, 2 lower) at about seven years.

3. Four lateral incisors (2 upper, 2 lower) at about eight years.

4. Four anterior bicuspids (2 upper, 2 lower) at about nine years.

5. Four posterior bicuspids (2 upper, 2 lower) at about ten years.

6. Four canines (2 upper, 2 lower) at about eleven years.

7. Four second molars (2 upper, 2 lower) at about twelve to fifteen years.

8. Four third molars (2 upper, 2 lower) at about seventeen to twenty-five years.

Abnormalities of the **permanent teeth**—

1. Increased vulnerability and brittleness, in divers grave constitutional affections, *e. g.*, rickets, profound anemia; in neglect and injury of the teeth, especially by escharotic drugs for cleansing of the teeth or medicinal purposes (*e. g.*, the tincture chloride of iron, acids).

2. Asymmetry, in harelip; cretinism and other forms of defective mentality; nasal obstruction; "mouth breathing"; thumb sucking.

3. Looseness, in gingivitis; ulcerative stomatitis; mercurialism; scurvy; pyorrhea alveolaris.

4. Hutchinson's teeth, *i. e.*, peg-shaped, dwarfed upper central incisors, notched in their cutting edge, in inherited syphilis.

5. Microdentism, small white transparent teeth, not abnormal, but occasionally seen in children of syphilitic parents.

6. Amorphism, tendency of teeth assuming abnormal shape (*e. g.*, incisor taking the shape of canine) frequently in syphilis, but also in nonsyphilitic children.

The **floor** of the **mouth** may present—

1. Adhesio linguæ, a frequent cause of difficult suckling; and later of difficult speech.

2. Sublingual ulcer, in protracted coughing, especially pertussis.

3. New growths, *e. g.*, ranula, fibroma sublinguale; in salivary calculi; inflammatory swelling.

The **palate** is—

1. Highly arched and asymmetrical, in divers forms of mental degeneracy; adenoids.

2. Defective or perforated, in congenital clefts of the palate; in syphilitic or gangrenous processes (*e. g.*, diphtheria, scarlatina).

3. Red, velvety, in scarlatina.

4. Punctiform or stellate, in measles or rötheln.

5. Vesicular with red areola, in chickenpox.

6. Papular, in smallpox.

7. Whitish-yellow eroded dots over the hamular process of the palate bone, in Bednar's aphthæ.

8. Minute, yellowish-white milia, in "epithelial pearls" (on both sides of raphé near the junction of the hard and soft palates).

9. White specks or scattered patches, in different forms of stomatitis.

10. Hemorrhagic and punctiform, in hemorrhagic diathesis; tuberculous and cerebrospinal meningitis; pernicious blood affections.

The **buccal mucous membrane** presents in addition to the discolorations occurring upon the palate, also the following:

1. Brownish, greenish or gray ulcer, in incipient noma.

2. Red spots with central, rounded, slightly elevated, bluish efflorescence (Koplik's spots), in measles.
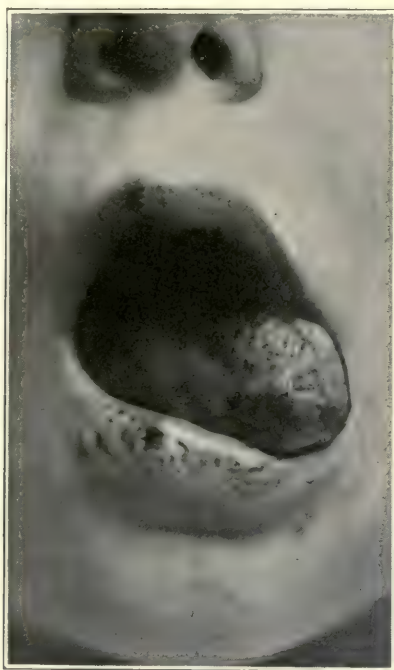
Fig. 10.—Ulcerative stomatitis involving also the lips and adjacent structures.

The **tongue** is—

1. *Large,* in congenital macroglossia; in cretinism; idiocy; glossitis.

2. *Furred,* in all acute and protracted forms of gastroenteritis; febrile diseases; nasopharyngeal catarrh.

3. *Red,* in scarlatina (strawberry tongue); stomatitis; glossitis; **gastritis** (hyperacidity).

4. *Yellow,* in biliousness; liver disease; chronic intestinal indigestion.

5. *Pale,* in anemia.

6. *Gray,* brown, and somewhat black, with red border and tip, in typhoid fever, in sepsis.

7. *Black,* in profound sepsis, in collapse impending death.

8. *Livid,* in general cyanosis; congenital heart disease; severe pneumonia.

9. *Spotted,* desquamating, in geographical tongue; hyperpyrexia; stomatitis.

10. *Fissured,* in glossitis desiccans; hyperpyrexia; burns.

11. *Ulcerated,* in severe forms of stomatitis; in syphilis; tuberculosis; traumatism (biting of the tongue during an epileptic fit; irritation by carious teeth).

12. *Dry,* in mouth-breathing; excessive thirst (*e. g.,* hyperpyrexia, diabetes) ; in sepsis.

13. *Protruding,* in macroglossia (*e. g.,* idiocy, cretinism).

14. *Drawn to one side,* in paralysis of the hypoglossal nerve (toward the diseased side) ; in peripheral facial palsy (toward the healthy side).

15. *Tremulous,* in hyperpyrexia; debility; chorea; disseminated lateral sclerosis; bulbar paralysis.

The **saliva** is—

1. Increased in quantity, in mercurialism; stomatitis; teething; idiotic conditions.

2. Diminished in quantity, in fever; from the effects of atropine, etc.; parotitis; glossitis.

The **uvula**—

May be elongated; the seat of a deposit which may extend from the tonsils or from the buccal mucous membrane (*e. g.,* stomatitis).

The **tonsils** are—

1. Enlarged, in divers forms of amygdalitis; diphtheria; scarlatina; pharyngitis; influenza; rheumatism; abscess; traumatism; glandular fever; foreign bodies (*e. g.,* calculi) ; new growths (*e. g.,* fibrous polypus, hydatid cyst).

2. The seat of a deposit, in follicular tonsillitis (small isolated white pellicles which coalesce) ; in parenchymatous tonsillitis (at first white, later yellowish green, resembling ''point of abscess'') ; in tonsillitis herpetiformis (vesicular deposit, ending in ulcer) ; in necrotic tonsillitis (yellowish-green patch) ; in influenza and pharyngitis (superficial exudation) ; in scarlatina and diphtheria (large pseudomembrane) ; in stomatitis mycotica (flour-like deposit).

In doubtful cases it is imperative to examine a smear of the tonsillar deposit microscopically or bacteriologically.

The Neck

The **lymphatic glands** are—

Enlarged, in all forms of angina, especially that due to diphtheria or scarlet fever; in affections of the mouth (*e. g.*, stomatitis, gingivitis) ; in parotitis; mastoiditis; rubella; glandular fever; pseudoleukemia; scrofulosis (tuberculosis) ; eczema capitis; local infections; nasal affections.

The **thyroid gland** is—

1. Enlarged, in goiter, exophthalmic goiter; endemic goitrous cretinism; thyroiditis; temporarily, before menstruation.

2. Atrophied or absent, in sporadic cretinism.

Tumefactions (other than those of the glands of the neck)—

1. Hematoma of the sternocleidomastoid, in the center or at sternal insertion of the sternocleidomastoid muscle.

2. Hygroma cysticum, between lower jaw and clavicle, attains enormous size.

3. Fistula colli congenita, at sternoclavicular articulation.

Pulsation of the—

1. Arteries, in heart disease; hyperpyrexia.

2. Veins, especially in tricuspid insufficiency.

Stiffness of neck (See "Attitude of Head," p. 118).

THE THORAX AND ITS CONTENTS

Auscultation and Percussion

Auscultation is best performed by a small biaural stethoscope, since with this instrument every inch of the infantile thorax can be thoroughly examined and small circumscribed lesions readily detected.

Normally the respiratory sound is puerile (rough vesicular) in infancy or early childhood; and vesicular in older children.

In auscultating the infantile *lungs* we should remember the following peculiarities: 1. During quiet respiration the inspiratory sound is fairly audible, while the expiratory sound is but slightly so; hence to obtain more distinct physical signs it is of advantage to disturb the infant, or to make it cry. 2. Owing to the larger diameter of the right bronchus, the respiratory sounds are louder on the right side than on the left. 3. Pure bronchial breathing is often normally heard over the interscapular regions, especially to the right of the spinal column. 4. Adventitious sounds originating in the nasopharynx and larynx are frequently transmitted to the chest and may be misinterpreted as signs of pulmonary disease.

The normal pulmonary percussion note is clear, loud, and somewhat tympanitic. It is somewhat metallic, when the child cries; cracked-pot-like, over the right subclavicular region; somewhat dull over the areas overlapping the liver, heart and spleen.

Percussion of the infantile *lungs* should be practiced while the patient is held in a sitting posture (watch heart action!) perfectly still and as erect as possible. It should be performed gently, preferably during the height of inspiration and expiration. Every portion of the lungs should
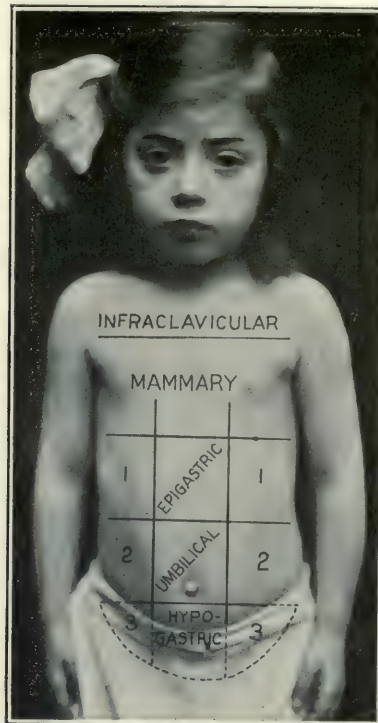
Fig. 11.—The thoracic and abdominal regions. *1.* Hypochondriac. *2.* Lumbar. *3.* Inguinal.

be carefully gone over, paying especial attention to the sub- and supra-clavicular spaces, which are not rarely the seat of consolidation, and the area corresponding to the tracheal bifurcation, which is often the seat of tuberculization of the bronchial glands. The physical signs are not always conclusive, if percussion is performed too forcibly (may give rise to covibration of the more distant parts); if the child cries (during the act of crying compression of the lungs by ascension of the diaphragm produces artificial dullness); if the position of the child is

faulty (*e. g.*, lying on the abdomen pushes the diaphragm upward and compresses the lungs); or if the thorax is bent sharply forward.

In auscultating the *heart* we should bear in mind the following: 1. Accentuation of the first sound is heard equally as well at the arterial and venous orifices. 2. Accentuation of the second sound is ordinarily not heard until about the age of puberty. 3. Both heart sounds are louder in children than in adults and are more widely transmitted. 4. Reduplication of the heart sounds is not uncommon, and generally the result of excitement. 5. In infants hemic murmurs are rare. 6.
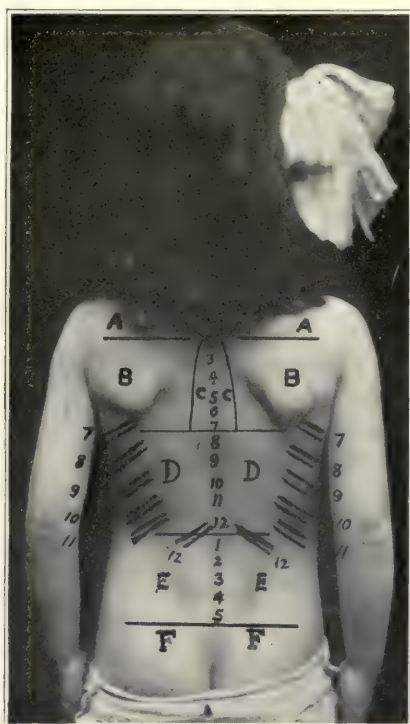
Fig. 12.—The regions of the back. *A*. Suprascapular or supraspinatus. *B*. Scapular. *C*. Interscapular. *D*. Infrascapular or lower dorsal. *E*. Lumbar. *F*. Sacral.

The heart beat, as to frequency and rhythm, is apt to undergo great variations on the slightest provocation.

Percussion of the child's *heart* should be performed very gently while the patient sits (watch heart action!) quietly and bent slightly forward. The data obtained on percussion while the child cries, holds its breath, etc., are not wholly to be depended upon, since during bodily unrest the heart is very apt to change its relation to the chest wall.

The same holds true in the event of the heart being overlapped by emphysematous lungs; or if the heart is left bare by atrophy of the adjacent lung portions, or by displacement or retraction of the heart or lungs by pleuritic or pericardial adhesions.

THE THORAX

The normal infantile thorax is round and somewhat cylindrical, its sagittal and transverse diameters being nearly equal. As the child grows older, the chest assumes a more conical shape, until, at puberty, it resembles that of the adult. The chest wall of the child is thin, elastic and yielding, owing to incomplete development of the muscular and bony structures. The ribs of the infant are nearly horizontal. The **measurements** of the thorax are—

In the newly born infant, about 13½ inches.
At one year, 18 inches.
At three years, 20 inches.
At six years, 23 inches.
At twelve years, 26 inches.

At the end of the fifteenth year, the measurement of the circumference of the chest is about half of that of the body length.

Up to about eighteen months the circumference of the chest nearly equals that of the head. If from the end of the second year on the circumference of the head exceeds that of the chest, there is a strong suspicion of hydrocephalus, marked rachitis and contraction of the chest through pulmonary disease or imperfect development (adenoids). On the other hand, if the chest measurement in early childhood by far exceeds that of the head, it is indicative either of an abnormality of the chest, e. g., distention by fluids, or of congenital maldevelopment of the head, e. g., microcephalus, infantilism.

Abnormal shapes of chest—

1. Barrel-shape (deep, short and broad), in emphysema, and the lung affections which precede it, e. g., asthma, pertussis; protracted laryngeal stenosis.

2. Flask-shape (flat, narrow and long), in phthisis pulmonum; nasopharyngeal stenosis, especially adenoids.

3. Funnel-shape (marked depression in lower portion of sternum), in rachitis; Barlow's disease; also congenital.

4. Pigeon- or chicken-breast-shape (protrusion of median portion of sternum and flattening of sides of chest), in rachitis; congenital heart disease.

5. Unilateral bulging, in pneumothorax; pleurisy or pericarditis with effusion; tumor; scoliosis (opposite side).

6. Unilateral flattening, in pleuritis retrahens (after absorption of fluid); pulmonary contraction, *e. g.*, tuberculosis; after pyothorax operation; scoliosis.

Tumefactions—

1. Costal, nodular, in rachitis (rachitic rosary); tuberculous and syphilitic processes; multiple exostoses.

2. Intercostal, doughy, in suppuration of the bronchial glands; empyema necessitatis; lung hernia.

3. Mammary, in mastitis; cold abscess; as a partial manifestation of parotitis; new growths.

Abnormal posture of scapulæ—

1. Prominent, uni- or bilaterally, "angel-wing" deformity, in congenital malformation; in emaciation. Unilaterally, in scoliosis; paralysis of the scapular muscles, *e. g.*, after local trauma; poliomyelitis; progressive atrophy.

2. Sunken, after empyema operation; in scoliosis.

Activity of the thorax in breathing—

1. Increased, bilaterally, in asthma; laryngeal obstruction; unilaterally, on the sound side, in pleurisy with effusion, pneumothorax; fixed deformities.

2. Diminished, bilaterally, in emphysema; hydrothorax; diffuse tuberculization; paralytic conditions of the chest wall; sclerema; collapse; unilaterally, in pleurisy with effusion; pneumothorax; pleurodynia; pleuropneumonia with "stitch pain."

Pain on **pressure—**

1. Superficial, in rheumatism of the chest muscles; intercostal neuralgia; affections of the ribs (caries, periostitis, fracture, etc.); localized abscesses (empyema necessitatis); and tumefactions (*e. g.*, mastitis).

2. Deep, in pleurisy; pneumonia; phthisis pulmonalis.

The Lungs[*]

The lungs are normally fully distended with air within the first few hours of life. In the premature or delicate infant full lung inflation may not occur until several weeks after birth. The lower lobes particularly may remain in a state of atelectasis.

The normal *boundaries* of the lungs differ somewhat with the age of the child. On both sides they project with their summits into the

---

[*]See "Auscultation" and "Percussion," p. 129.

supraclavicular fossæ. From here they descend in the following manner:

The **right lung** (lower border) lies—

In the sternal line at a point corresponding to the fifth (upper border) rib.

In the parasternal line at a point corresponding to the fifth (lower border) rib.

In the mammary line at a point corresponding to the sixth rib.

In the axillary line at a point corresponding to the seventh rib.

In the scapular line at a point corresponding to the tenth rib.

The **left lung** (lower border) lies—

In the sternal line at a point corresponding to the fourth rib.

In the parasternal line at a point corresponding to the fourth rib.

In the mammary line at a point corresponding to the sixth rib.

In the axillary line at a point corresponding to the seventh or eighth rib.

In the scapular line at a point corresponding to the tenth rib.

Posteriorly, the base of the left lung is slightly lower than that of the right lung.

Number of **respirations** per minute—

In the newborn, from 35 to 40.

At the end of the first year, 30.

At the end of the second year, 25.

At six years, 22.

At twelve years, 20.

Character of **respiration**—

1. Abdominal, in children under four years of age.

2. Costoabdominal, in children (male and female) up to ten years; in the male, in older ones.

3. Thoracic, in girls over ten years.

4. Regularity of respiratory rhythm is usually not fully established before the age of two years.

Abnormalities of **respiration**—

1. Increased frequency, in respiratory and circulatory diseases (see "difficult breathing"); pyrexia; emotional excitement; compression of the lungs by an accumulation of gas; fluids, or solid masses.

2. Diminished frequency, in grave central disease; extreme weakness; poisoning from belladonna, opium, etc.

3. Costal breathing in boys over ten years old, and increased costal breathing in girls, in inflammatory diseases of the abdominal and pleural cavities (by interference with the action of the diaphragm)
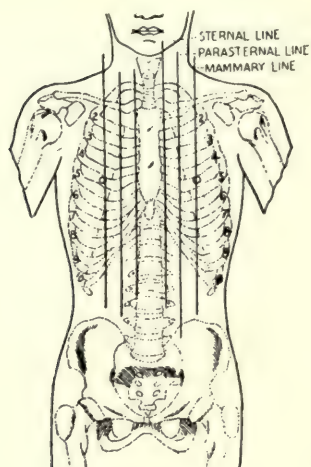
Fig. 13.—Diagnostic lines of the thorax.
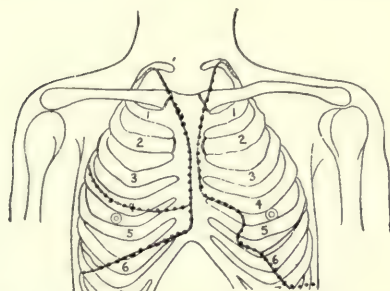
Fig. 14.—Anterior boundaries of the lungs.
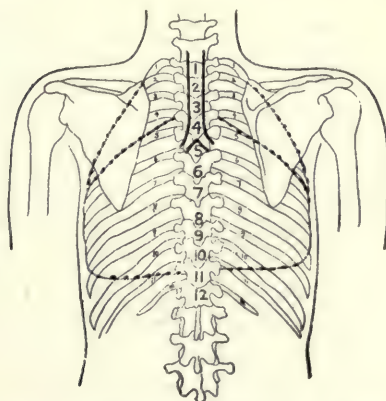
Fig. 15.—Posterior boundaries of the lungs.

e. g., peritonitis, pleuritis; in abdominal distention by gases, fluids, or solid masses; in paralysis of the diaphragm, *e. g.*, bulbar paralysis, polioencephalitis, neuritis (postdiphtheritic) of the phrenic nerve; in drug poisoning; in hysteria.

4. Purely abdominal breathing, especially in girls over ten years old, in emphysema; scleroderma; paralysis of respiratory muscles, *e. g.*, bulbar paralysis, poliomyelitis.

5. Irregular breathing, in conditions associated with "difficult breathing"; in cerebrospinal affections; in atelectasis; painful diseases of the respiratory muscles; in hysteria.

6. Stertorous breathing, in nasopharyngeal obstruction, *e. g.*, retropharyngeal abscess, adenoids; in uremic or apoplectic coma.

7. Cheyne-Stokes' breathing, occasionally in infants during sleep; in heart failure from divers causes; in meningitis, especially the tuberculous variety; in meningeal hemorrhage, tumors or abscess exerting pressure upon the brain; in drug poisoning, *e. g.*, opium; in death agony.

8. Difficult or labored breathing (dyspnea), in laryngeal, tracheal or bronchial obstruction from divers causes, *e. g.*, croup, diphtheria, large thymus, asthma, etc.; in affections associated with diminution of the usual pulmonary breathing area, such as active or passive congestion, *e. g.*, pneumonia, pleurisy or pericarditis with effusion, compression or displacement by neoplasms, deformities of the thorax, advanced pulmonary tuberculosis; in grave circulatory disturbance inducing deficient oxygenation of the blood or obstruction to pulmonary circulation, *e. g.*, blood or heart diseases ("cardiac asthma"); in conditions giving rise to "irregular breathing" (*q. v.*), "stertorous breathing" (*q. v.*), and "Cheyne-Stokes' breathing" (*q. v.*,), in neuroses, *e. g.*, hysteria, neurasthenia—asthma hystericum.

Abnormal respiratory sounds—

1. Vesicular, exaggerated, in bronchial inflammation; atelectasis.

2. Weak, in thickened pleura; moderate pleuritic effusion; emphysema.

3. Absent, in extensive pleuritic effusions.

4. Bronchial, over the seat of the lesion, in pneumonia; tuberculization; above the seat of lesion, in compression of the lung by tumors in the chest cavity or pleuritic exudates.

5. Amphoric, in smooth-walled cavities; open pneumothorax.

Abnormal secretory sounds—

1. Dry, sibilant and sonorous rhonchi, in bronchitis; asthma (wheezing and whistling).

2. Dry, crackling, in incipient phthisis (apex); beginning of second stage of pneumonia.

3. Moist, large and medium-sized râles, in bronchitis (larger bronchial tubes) with abundant secretion; in cavities.

4. Moist, small râles, in capillary bronchitis.

5. Moist, crepitant (fine) râles, in croupous pneumonia (crepitatio indux or redux; catarrhal pneumonia; capillary bronchitis (in conjunction with coarse râles); tuberculization; pulmonary edema (in conjunction with larger moist râles).

6. Metallic tinkling, in pneumothorax.

7. Metallic splashing or gurgling, in sero- or pyopneumothorax.

8. Friction sound, in pleuritis sicca; pleuropneumonia; miliary tuberculosis. It is not altered by coughing, as is the case with râles.

Vocal resonance*—

1. Diminished, in bronchitis with free secretion; pleurisy with effusion; obstruction of bronchial tubes; emphysema; pneumothorax.

2. Increased, in tuberculization; pneumonia (over consolidation).

3. Bronchophony (concentration of voice near the ear), in tuberculization; pneumonic consolidation; compressed lung above pleuritic effusion; bronchial dilatation.

4. Exaggerated bronchial whisper; the same as for bronchophony $(q. v.)$.

5. Pectoriloquy (complete transmission of sound), the same as for bronchophony $(q. v.)$.

6. Amphoric voice ("the echo"), in large cavity; pneumothorax.

7. Egophony, bleating (goat-like resonance of voice), in pleurisy with effusion (near upper boundary of dulness); pleuropneumonia; hydrothorax.

Abnormal percussion resonance—

1. Dull, or diminished resonance, in pneumonia; tubercle; neoplasms; pulmonary gangrene; pulmonary abscess with thick masses; pleuritic thickening; atelectasis.

2. Flat or absence of resonance, in pleurisy with effusion; hydrothorax; hemothorax. Resonance may alter with change of patient's position. Also in last stage of pneumonia with extensive consolidation.

3. Tympanitic, or drum-like, resonance, in tuberculosis (cavities); open pneumothorax; lung atrophy; above pericardial or pleuritic exudations or near neoplasms—the result of increased air pressure; pulmonary edema; moderate emphysema.

4. Amphoric, metallic, or concentrated tympanitic sound, in large

---

*Vocal resonance elicited on auscultation corresponds to vocal fremitus as obtained by palpation. Fremitus is increased in consolidation, and diminished in effusions.

tuberculous cavity with solid and tense walls lying close to the chest wall; occasionally heard in healthy child during crying.

5. Cracked-pot resonance, in pulmonary cavity communicating with the bronchial tubes—usually in tuberculosis; may be elicited also in healthy child during talking or singing.

6. Bandbox note (abnormally loud and deep), in pronounced emphysema; pneumothorax with strong tension of the chest wall.

Cough

It is essentially a reflex act arising from direct or indirect irritation of the respiratory center. In a measure it can be voluntarily produced or suppressed. The ability to cough is lost in paralysis of the cricoarytenoid or the respiratory muscles; hence cessation of coughing—with plenty of mucus in the bronchial tubes—particularly in pulmonary disease, is considered a bad omen. The nature of the cough may often be decided upon from its character.

The **cough** is usually—

1. Short and somewhat hoarse, in nasopharyngeal catarrh and adenoids.

2. Loud and barking, in laryngitis and spasmodic croup.

3. Dull, barking and somewhat moist, in ulceration of the larynx (diphtheria, syphilis, etc.).

4. Dry, tight and whistling, in early bronchitis.

5. Soft, deep, and loose, in advanced bronchitis.

6. Paroxysmal and whooping, in pertussis and other spasmodic affections; tuberculosis of the bronchial glands.

7. Hemming, in incipient phthisis and in nervousness.

8. Short, sharp and painful, in pneumonia, pleurisy, and cardiac disease.

9. Deep and distressing, in chronic phthisis, asthma, emphysema, etc.

Too much reliance should not be placed upon the character of the cough, as it is very apt to vary with the duration of the cough, medication and complications. By far more reliable information can be obtained from a careful examination of the expectoration.

Sputum, Expectoration

In cases where the children cannot or will not expectorate, the sputum may be obtained by introducing into the throat a sterile cotton swab or fenestrated stomach tube—both of which usually re-

ceive enough of sputum during the act of coughing to suffice for ordinary examination.

The **expectoration** is—

1. Mucous, frothy, grayish-white, in acute catarrh of the air passages.

2. Mucopurulent, tenacious, yellowish-gray, in chronic tracheobronchial catarrh; in pertussis (voluminous, often mixed with vomitus); in asthma (Curschmann's spirals, Charcot's crystals); in bronchiectasis (periodic "mouthful expectoration," separable into a purulent and mucoserous layer).

3. Purulent, fetid, dirty grayish-green, in fetid or putrid bronchitis (separable into three layers; suspended in the lowest, purulent layer are Dittrich's plugs); in pulmonary abscess (separable into two distinct layers, containing a great number of micrococci, elastic fibers, fat crystals, etc.); in pulmonary gangrene (same as putrid bronchitis, plus tissue fragments).

4. Serous, prune-juice-like, and profuse, in pulmonary edema.

5. Bloody, in nasopharyngeal catarrh with violent paroxysms of coughing (occasional streaks of blood); in foreign bodies in the air passages (bright red mixed with frothy mucus); in pneumonia (uniformly stained, "rusty" sputum to dark "prune juice" color with pneumococci); in influenza (often bright red and profuse); in heart disease with edema (the same as in pulmonary edema from other causes; besides "heart-cells"); in tuberculous lesions of the air passages (either large hemorrhage, "hemoptysis," or blood stained "nummular" and heavy sputum, containing tubercle bacilli); in neoplasms ("red currant"-like sputum, with characteristic histologic structures); in vicarious menstruation; hemorrhagic diathesis, and hysteria. (See "Hematemesis" and "Epistaxis.")

The expectoration contains numerous microorganisms and occasionally bile (icterus), hydatid hooklets, distomum pulmonale, and cercomonas.

The Heart*

The heart is comparatively larger in infancy than in later life. It is relatively largest at birth, and smallest at about the age of seven years. At birth the walls of both ventricles are nearly of equal thickness, but as the infant grows older, the left ventricle rapidly gains in thickness, so that by the end of the second year it is almost twice as thick as the right ventricle.

*See "Auscultation" and "Percussion," p. 129.

Corresponding to the relatively larger size and more transverse position of the heart of the young child, its boundaries are greatly at variance from those of the heart of the adult.

The **boundaries** of the normal heart—

The *apex beat is situated*—

To the left of the mammary line, in the fourth intercostal space, up to the fourth year of age.

At the mammary line, slightly below the fifth rib, up to the eighth year.
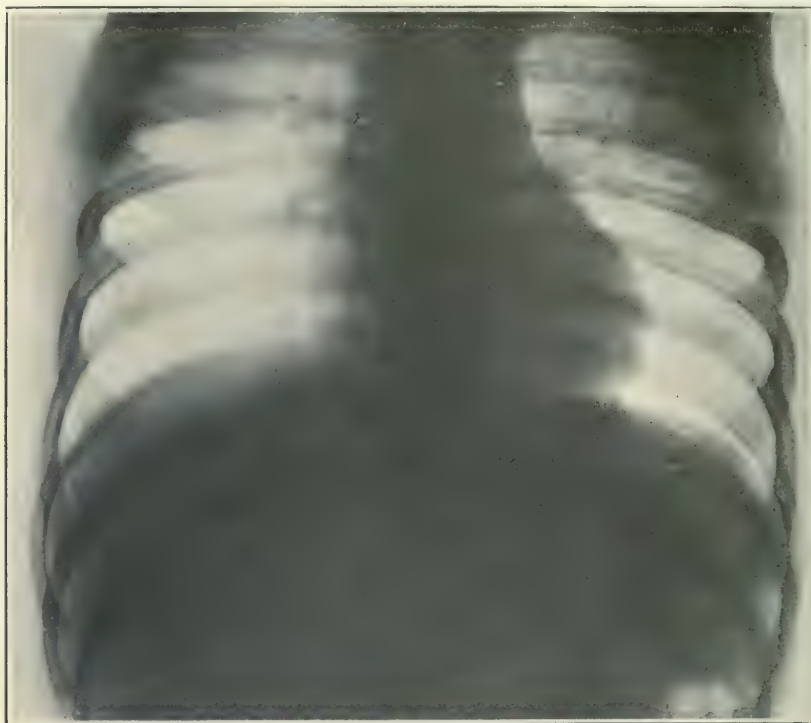
Fig. 16.—Normal heart of a child three years old.

Slightly to the right of the mammary line, in the fifth intercostal space, up to the twelfth year.

Between the mammary and parasternal lines, *i. e.,* the same as in the adult, in children over twelve years.

The *Relative "heart dulness"* in infants is bounded as follows:

Above, by a line corresponding to the lower border of the second rib.

On the left side, by a line parallel and slightly to the left
of the left mammary line.

On the right side, by the right parasternal line.

Below, by a somewhat semicircular line along the fifth rib.

As the child grows older and the heart assumes a more oblique and
lower position, the boundaries of the relative heart dulness gradually
fall in line with those of the adult.

The *absolute "heart dulness"* in infants is bounded as follows:

Above, by the upper border of the fourth rib.

On the left side, by the left mammary line (slightly to the
right of it).
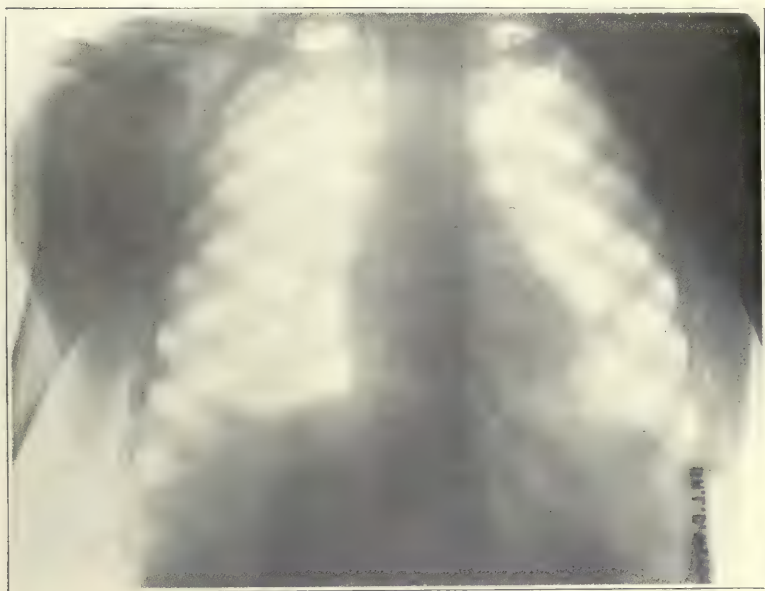
Fig. 17.—Normal heart of a child eight years old.

On the right side, by the left sternal line.

Below, by a line corresponding to the upper border of the
fifth rib.

These boundaries, like those of the relative heart dulness, change
gradually with the advance of the child's age, so that in children over
twelve years old, the upper boundary is formed by the fourth rib; the
lower by a line drawn parallel to and between the fifth and sixth ribs;
on the right side, by the sternal line; and on the left by a line midway
between the parasternal and mammary lines.   (See Figs. 18, 19, and 20.)

The normal **pulse rate** (most reliable when patient is asleep)—
 In the newborn from 120 to 150 per minute.
 At one year old, 100 to 120 per minute.
 At four years, 90 to 100 per minute.
 At eight years, 80 to 90 per minute.
 At twelve years, 75 to 80 per minute.
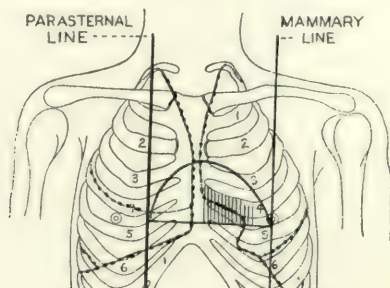
Fig. 18.—Up to four years.
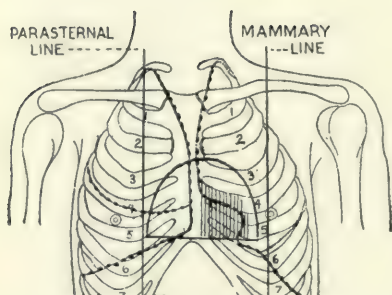
Fig. 19.—Up to eight years.   Fig. 20.—Up to twelve years.
The relative and absolute heart dulness at different ages.
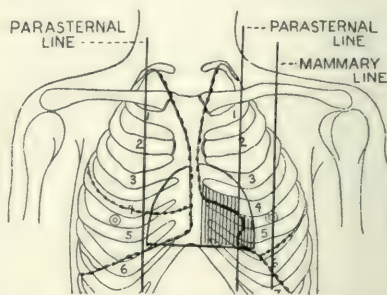
Normal **pulse respiration** ratio is approximately 1:4. A ratio of 1:3 or
 less is a certain indication of pulmonary disease, especially pneu-
 monia.

Apex beat—
 1. *Displaced—*
  Outward, to the left, in hypertrophy of the right ventricle;
   dilatation of the right ventricle; right-sided pleurisy with
   effusion; right-sided pneumothorax; abdominal distention
   pushing the diaphragm upwards and the heart to the left.
  Outward and downward, in hypertrophy of the left ventri-
   cle; dilatation of the left ventricle; pericardial effusion;
   congenital or acquired (by pressure from above, *e. g.*, tu-
   mor or abscess) dislocation of the heart.

Inward, to the right, in left-sided pleuritic effusion; pronounced left-sided deformity of the thorax; persistence of the embryonic position or *situs inversus* (up to dextrocardia).

2. *Effaced* (*i. e.*, apex beat is invisible and barely palpable), in obesity; pericardial effusion; heart failure; emphysema; edema cutis; tumors.

3. *Diffuse and weak* in irregularity of the heart associated with grave heart disease.

4. *Diffuse and strong,* in cardiac hypertrophy; hyperpyrexia; overstimulation; excitement. The cardiac impulse may only *appear* strong when the chest wall is very thin.

Heart sounds—

Accentuation of—

1. Systolic mitral, in excitement; fatigue; fever; hypertrophy of the left ventricle.
2. Diastolic pulmonic, in hypertrophy of right ventricle.
3. Diastolic aortic, in hypertrophy of left ventricle.

Weakening of—

1. Systolic mitral, in dilatation of the left ventricle; loss of compensation.
2. Diastolic pulmonic, in dilatation of the right ventricle (*e. g.*, relative tricuspid insufficiency); stenosis of pulmonary artery.
3. Diastolic aortic, in aortic stenosis.

Division (double) of diastolic at apex, in mitral stenosis; adhesive pericarditis.

Gallop rhythm, in heart failure from various causes (*e. g.*, incipient diphtheritic paralysis); noncompensating heart disease; tachycardia.

Metallic ringing, in pneumopericardium; pneumothorax; large pulmonary cavity; intense meteorism.

Heart murmurs—

1. Systolic, loudest at apex and transmitted to axilla and angle of left scapula, in mitral regurgitation.
2. Systolic, loudest at base (midsternum) and transmitted to the arteries upward and sometimes over the whole sternum, in aortic obstruction.
3. Systolic, at base, but *not* transmitted upward, in pulmonic obstruction.
4. Systolic, loudest at ensiform cartilage, in tricuspid regurgitation.

5. Diastolic, loudest at base, and transmitted to apex and ensiform cartilage, in aortic regurgitation.

6. Diastolic, or presystolic, loudest at apex, in mitral obstruction.

7. To-and-fro-friction, superficial, limited to precordium; not influenced by respiration (as is the case in pleuritis sicca), in fibrinous pericarditis.
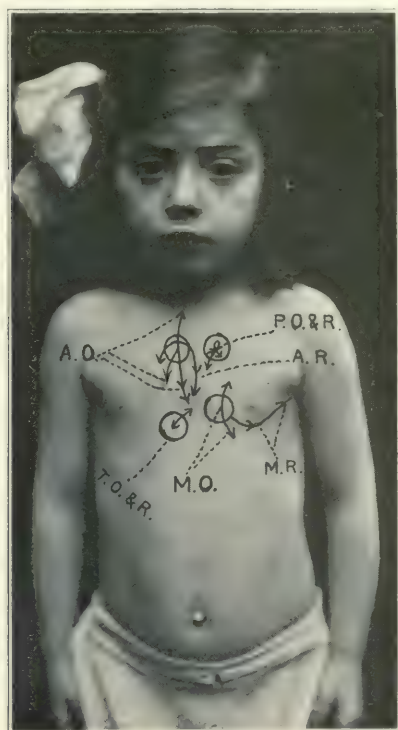
Fig. 21.—Topography of cardiac valves. Points of transmission of heart murmurs. *A. O.* Aortic obstruction. *P. O.* and *R.* Pulmonic obstruction and regurgitation. *A. R.* Aortic regurgitation. *T. O.* and *R.* Tricuspid obstruction and regurgitation. *M. O.* Mitral obstruction. *M. R.* Mitral regurgitation.

Areas of heart dulness—

Enlarged—

1. To the left, in hypertrophy or dilatation of the left ventricle.

2. To the right, in hypertrophy or dilatation of the right ventricle.

3. Bilaterally, in pericardial effusion. The area of dulness is larger in sitting than in recumbent posture; it is often triangular; wider below than above.

Reduced—
> In pulmonary emphysema; pneumopericardium.

Displaced—
> 1. In congenital malpositions, *e. g.*, dextrocardia, mesocardia, diaphragmatic hernia.
> 2. In acquired affections, such as pneumothorax; pleurisy with effusion; neoplasms; pleuritic retraction; atrophy of the lungs.

The pulse—
> 1. Frequent, in fright; excitement; fear; febrile diseases (except uncomplicated typhoid or meningitis); valvular heart diseases (except aortic stenosis); anemias, especially on slight exertion; tachycardia; exophthalmic goiter; convalescence from acute affections; paralysis of the heart (central or peripheral paralysis of pneumogastric nerve); heart failure (*e. g.*, collapse in febrile diseases).
> 2. Slow, in uncomplicated typhoid fever or meningitis; after crises (*e. g.*, pneumonia); acute nephritis; catarrhal jaundice; intracranial pressure (*e. g.*, hydrocephalus, hemorrhage, tumors); heart disease, such as aortic stenosis, myocarditis; bradycardia; profuse hemorrhage; marked inanition (*e. g.*, a pyloric stenosis); opium poisoning.
> 3. Irregular, in last stages of valvular heart disease; myocarditis; profound anemia (on exertion); nervous palpitation; indigestion (flatulent colic).

In the **irregular pulse** we distinguish the—
> 1. *Intermittent pulse—*
> Pulsus alternans (every second beat weak).
> Pulsus bigeminus (every third beat weak).
> Pulsus trigeminus (every fourth beat weak).
> 2. *Intercidens pulse* (several regular beats suddenly followed by a small beat and pause), in heart weakness.
> 3. *Paradoxic pulse* (the pulse grows smaller or ceases entirely on deep inspiration), in adhesive pericarditis; constriction of the air passage; mediastinal tumors; during ''whoop'' in pertussis.
> 4. *Dicrotic or double pulse* (in part explained by a loss in the muscular tone in the arteries, so that the arterial impulse is separated from that of the ventricles by a perceptible interval), in typhoid fever and less marked in other acute febrile diseases; in chronic wasting diseases, especially tuberculosis; in anemias; after great loss of blood.

5. *Asymmetric (radial) pulse,* in congenital anatomic variations of the artery on one side; acquired narrowing, compression; or cicatricial contraction of the radial, brachial, axillary, subclavian or innominate artery; aneurysm of the aforementioned arteries or of the aorta; in pneumothorax compressing the subclavian artery.

THE ABDOMEN AND ITS CONTENTS

In order to save time, inspection and palpation of the abdomen may at once be supplemented by percussion, succussion, etc. To judge
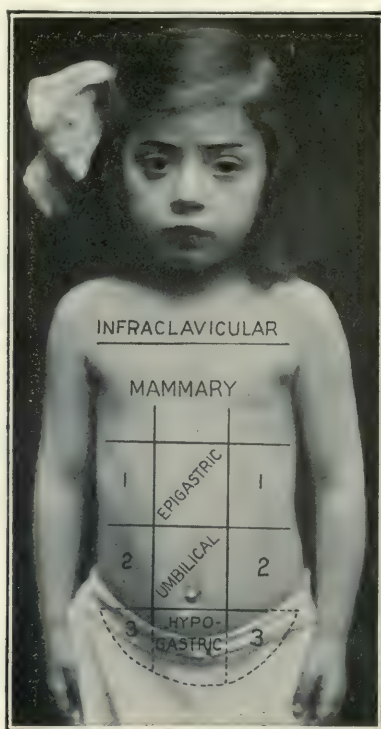
Fig. 22.—The thoracic and abdominal regions. *1.* Hypochondriac. *2.* Lumbar. *3.* Inguinal.

matters correctly we should bear in mind the normal relations of the abdominal parietes to the underlying structures.

The **abdominal wall** is moderately arched; readily compressible without undue resistance or pain; moves slightly upward and downward quite evenly and regularly with inspiration and expiration; and on

percussion yields a loud, tympanitic sound over all portions of the abdomen engaged by the intestines.

The **stomach** at birth is nearly cylindrical and lies obliquely in the abdominal cavity. Thus, the cardiac end on a level with the tenth dorsal vertebra, and the pyloric end in the median line and slightly to the right, midway between the tip of the xiphoid cartilage and the umbilicus. The pylorus is not palpable. Gradually the fundus increases in size (at seven months it is twice the original length) and the stomach assumes a transverse position in such a manner that five-sixths of its volume occupies the left half of the abdomen and one-sixth the right. The capacity of the stomach varies, of course, with the age and size of the child, as fully given when discussing ''infant feeding'' p. 55. The stomach empties itself in breast fed babies in two hours; in artificially fed in three hours. The stomach is dilated in congenital pyloric stenosis (shown by the bismuth radiogram test, see Fig. 58); also in general atony.

The infantile **intestines,** especially the small intestine, are relatively longer than those of the adult. At birth the small intestine is about 9 feet long, the large intestine about 18 inches, the sigmoid flexure forming about half of the colon. The capacity of the infantile intestines is relatively greater than in the adult, but their musculature is thinner and weaker, hence the tendency to constipation and colic.

The **intestines** are—

1. Dilated, in megacolon congenitum; above constriction in stenosis or atresia; intussusception; chronic constipation; prolonged meteorism.
2. Contracted, below the seat of constriction; compression by abdominal tumors.

The **liver** of the newborn is relatively very large in size, much larger than in the adult, constituting in the former about one-eighteenth, and in the latter about one-thirty-sixth of the entire body weight.

As the child grows older the size of the liver is greatly reduced, but owing to the sloping course of the lower ribs the liver appears considerably larger than it actually is.

Normal **boundaries** of the **liver** (as determined by percussion)—

1. Upper border, at midsternal line, base of ensiform cartilage; mammary line, sixth rib; midaxillary line, eight rib; scapular line, tenth rib.
2. Lower border, parasternal line, seventh rib; mammary line, about ½ inch below free border of the ribs; midaxillary line, tenth rib; scapular line, eleventh rib.
3. Left border, joins lower absolute heart dulness.

4. Right border, joins the right kidney.

Its position varies greatly with the ascent and descent of the diaphragm—rises with expiration and descends with deep inspiration. In the same manner it rises with intestinal meteorism and descends with overdistention of the lungs through disease, *e. g.*, emphysema or pneumothorax.

Fig. 23.—Dissection of still-born child. Note the relatively large size of the liver; note also peculiar course of sigmoid. (Henry Enos Tuley.)

The **liver** is—

1. Enlarged, in congenital syphilis; tumors; or cysts; liver abscess; chronic heart disease; acute septic processes; abdominal tuberculosis; splenomegaly (Gaucher); amyloid degeneration; hypertrophic cirrhosis; Banti's disease.

2. Displaced, in congenital dislocation; rachitis; right extensive pleural effusions.

The **spleen** lies in close contact with the diaphragm, and extends from the left midaxillary line to a point near the left border of the spinal column. Its upper border follows the ninth rib, its lower border the eleventh rib, for the most part bounding the left kidney. Normally the spleen cannot be outlined by percussion, but during deep inspiration it can sometimes be palpated at the free borders of the tenth and eleventh ribs.

Fig. 24.—Topography of the liver and spleen.

The **spleen** is—

1. Enlarged, primarily, in leukemia, pseudoleukemia; in von Jaksch's anemia; splenitis; splenomegaly (Gaucher); Banti's disease; tumors; secondarily, in malaria; all septic processes; tuberculosis; typhoid; rachitis; syphilis; liver disease.

2. Displaced, in pleural effusions; deformities of chest; after severe coughing (pertussis).

The **kidneys** are situated upon the right and left sides of the spinal column, and extend from the levels of the twelfth dorsal to the second lumbar vertebræ. The uppermost end of the right kidney

(the suprarenal capsule) is slightly overlapped by the liver; that of the left kidney by the spleen. Normal kidneys are usually palpable when the abdomen is relaxed, but can never be outlined by percussion.

The *urinary bladder* is situated underneath the symphysis pubis, but when fully distended rises above it, eliciting dull percussion resonance.

Abnormal size and **shape** of the **abdomen**—

1. Large and uniform, in flatulence; in acute and chronic gastroenteritis; acute peritonitis from various causes; late stage of grave pneumonia; intestinal atony or paralysis; extensive ascites.

Fig. 25.—Topography of kidneys, spleen, and liver. *S.* Spleen. *L.* Liver. *K.* Kidneys.

2. Retracted, in collapse, especially from gastrointestinal disease; in inanition (pyloric or esophageal stenosis); meningitis ("scaphoid abdomen"); general cachexia and loss of fat and muscle.

Increased abdominal resistance—

1. Local, in localized affections of the different abdominal organs (tumors, abscesses, foreign bodies, *e. g.,* fecal impaction; helminthiasis).

2. General, in hyperesthesia; rheumatism of abdominal muscles; colic; peritonitis from different causes; appendicitis; sclerema; scleredema; extensive dropsical effusion.

Abdominal pain—

In all conditions enumerated under "abdominal resistance," except sclerema; scleredema, and dropsy. In pneumonia, pleurisy (reflex); in cholelithiasis; gastralgia; ulcer; nephrolithiasis; cystitis; vesical calculi; intestinal adhesions; ren mobilis; uterine and ovarian diseases (in older girls); in hysteria.

Visible intestinal peristalsis—

1. Normal, in very thin and lax abdominal parietes, *e. g.*, congenital diastasis recti abdominis (Fig. 37); infantile athrepsia; atrophy due to paralysis.
2. Abnormal (increased or reversed), in pylorus stenosis; intestinal obstruction or constriction from various causes; congenital dilatation of the colon.

Palpable or visible herniæ—

1. In the linea alba (ventral; diastasis recti abdominis).
2. At the umbilicus (congenital hernia of the cord, ectopia viscerum; simple umbilical hernia).
3. In the lumbar triangles (lumbar hernia; lateral ventral hernia).
4. In the inguinal regions (direct and oblique inguinal hernia).
5. At the femoral fossa (femoral or crural hernia).

The Diagnostic Significance of Chronic Abdominal Enlargement

Chronic abdominal enlargement in children is of common occurrence and in the majority of instances is due to *rachitis* and protracted intestinal indigestion. Occasionally, however, it is the result of certain grave intraabdominal pathologic conditions.

The *liver*, occupying as it does a wide area of the upper abdominal cavity (see Fig. 23), is very prone to cause considerable abdominal enlargement even when slightly exceeding its normal boundaries, as in slight downward displacement or enlargement. Displacement of the liver, which, by the way is often mistaken for enlargement, is usually the result of rachitic deformity of the chest, but may occasionally be met with in consequence of large pleuritic effusions, emphysema, and pneumothorax. In rachitic displacement the diagnosis can readily be made by percussion, when it is found that the liver dulness, instead of beginning on a level with the sixth rib, starts anywhere below this upper normal boundary. The same holds true with displacement accompanying emphysema or pneumothorax,

but here we have in addition the clinical signs of these affections to go by, especially barrel-shaped chest and exaggerated resonance on percussion in the former, and the acute onset and tympanitic percussion sounds in the latter. The diagnosis of hepatic displacement secondary to pleurisy with effusion is often difficult, owing to the difficulty of distinguishing the dulness of the liver from that of the pleuritic effusion, but the diagnosis can ordinarily be cleared up by exploratory puncture. Enlargement of the liver sufficient to produce marked abdominal enlargement is usually observed in connection with syphilis, neoplasm, abscess, hepatic cyst, and congenital obliteration of the bile duct, or secondary to pronounced heart or spleen affections. In older children we must think also of pericarditic pseudocirrhosis of the liver (Pick's disease), which is associated with rheumatic or tuberculous obliteration of the pericardium and is manifested by enlargement of the spleen and liver and by ascites. In older children also we occasionally meet with abdominal enlargement due to ascites associated with hypertrophic and atrophic cirrhosis of the liver owing to abuse of alcoholic beverages.

Occasionally a phantom tumor (localized meteorism and contraction of the intestinal muscles; usually of a hysterical nature) in the epigastrium, may be mistaken for a large liver. The tumor gives a tympanitic note; there is no fluctuation; it disappears under anesthesia.

In examining the *spleen* we should bear in mind that any spleen that is palpable is either diseased or displaced. The displacement may be either congenital, the so-called "wandering spleen," or acquired as a result of prolonged and severe coughing, *e. g.*, in pertussis. A displaced spleen is rarely the cause of marked abdominal enlargement, and the same is true of a slightly enlarged spleen, unless it be associated with rachitis. The spleens, large enough independently greatly to influence the abdominal contour of children, are ordinarily encountered with anemia pseudoleukemica infantum, leukemia, syphilis, neoplasms or primary splenohepatomegaly (Gaucher) (see Fig. 158), more especially with the latter affection. Finally, on very rare occasions abdominal enlargement is found to be due to so-called Banti's disease, which is characterized by splenomegaly, anemia, cirrhosis of the liver, ascites, and hemorrhages.

The usual *kidney* affections are not productive of abdominal enlargement except in their late stages as a result of dropsical effusions within the abdominal cavity, or of secondary involvement of other organs. In such cases the diagnosis is obvious. As we palpate the kidneys, which procedure is readily accomplished especially when they are displaced or enlarged, we should be watchful for hydroneph-

rosis and neoplasms. Hydronephrosis, in order materially to change
the outline of the child's abdomen, is usually large enough readily to
be felt as an immovable, fluctuating mass in the lumbar region, but
considerable difficulty is experienced in differentiating unilateral hy-
dronephrosis from a cystic tumor of the kidney. As hydronephrosis is
due either to congenital atresia or acquired occlusion of the ureter, its
differentiation from cystic kidney can be made only by a careful ure-
teroscopic examination or puncture of the mass (showing the pres-

Fig. 26.—Sarcoma of the left kidney.

ence of urine) through the abdominal wall. Ordinarily one would
rarely err in diagnosing a kidney neoplasm rather than hydronephro-
sis, since the former is by far more common than the latter. This
is true, especially of sarcoma. Hard tumors of the kidney are best
diagnosed by palpation, if need be under anesthesia, although some
diagnostic help is also obtained from the x-rays. No great reliance
should be placed upon hematuria as a characteristic sign of renal

neoplasm, since blood in the urine is frequently found in renal tuberculosis, hemorrhagic nephritis, purpura, and other diseased conditions, and is often absent in kidney tumors when large enough to obstruct the ureter so that no urine is excreted from the affected side.

Next to rachitis, *tuberculosis* of the peritoneum or intestines forms the most frequent cause of abdominal enlargement in children. But while we meet these cases almost daily in hospital, dispensary, and private practice, the diagnosis is not always easy. It is often especially difficult to detect tuberculosis of the intestines. The tuberculous lesions are usually located in the lower portions of the ileum,

Fig. 27.—High degree of rachitis. Abdominal enlargement chiefly in epigastric region.

ileocecal region, and colon, but owing to the accompanying intense meteorism, the intestinal tumefaction is beyond reach of palpation, particularly during the early stages. However, as the diagnosis can frequently be established by the demonstration of tubercle bacilli in the stools, these should always be subjected to minute bacteriologic examination whenever stubborn diarrhea and rapid emaciation prevail. Less difficulty, as a rule, is experienced in the diagnosis of tuberculous peritonitis, because the tuberculous peritoneal masses are more superficial and hence more readily palpable, and also because of the pres-

ence of fluid in the abdominal cavity. A positive tuberculin reaction, of course, is corroborative of the diagnosis. It should be borne in mind, however, that a negative result by no means proves the absence of tuberculosis. This point is deserving of special emphasis. I desire to call particular attention to a physical sign which proved to me very helpful in differentiating abdominal enlargement associated with rachitis from that of tuberculous peritonitis. Whereas in rachitis (Fig. 27) the greatest prominence of the abdomen is manifested at the epigastrium, in tuberculous peritonitis (Fig. 28) the abdominal circumference is largest at or below the umbilicus (hypogastrium). This differential physical sign can best be elicited by careful measurements

Fig. 28.—Tuberculous peritonitis. Abdominal enlargement most marked in hypogastric region.

of the abdominal circumference by means of a tape measure, but can readily be determined also by mere inspection. This sign can be explained by the fact that in tuberculous peritonitis the inflammatory exudate accumulates at the bottom of the abdominal cavity and thus distends the surrounding abdominal wall. To make correct use, however, of this sign we must be sure to exclude large dermoid cysts of the ovary and an overdistended bladder, both of which conditions are apt to lead to diagnostic errors.

There is one other intestinal abnormality which often gives rise to an enormous abdominal enlargement in children, and that is, congenital or acquired *hypertrophy and dilatation of the colon,* the so-called Hirsch-

sprung's disease (see Fig. 34). While it was originally thought to be a congenital affection only, it has lately been shown to develop gradually also after birth. In these cases particularly the diagnosis is often very difficult, but nowadays, with the help of the x-ray, the diagnosis can readily be made, even if the usual symptoms of the disease fail to disclose the pathologic condition.

Vomiting—

1. Gastroenteric (associated with nausea and effort; followed by relief), in simple gastroenteric disturbances and intoxication; pyloric stenosis or spasm; acidosis; intestinal obstructon from various causes; appendicitis; peritonitis; the effect of emetics or poisonous drugs (taken by mouth).
2. Cerebral (explosive; watery, recurrent without relief).
   (a) Direct, in acute and chronic affections of the cerebrospinal system; shock; psychic emotion.
   (b) Reflex, in extracranial irritation of the cranial nerves, e. g., of the optic or oculomotor nerves in visual defects; of the auditory nerve, in otitides; pneumogastric, in pulmonary and cardiac diseases. Also in toxemia, by bacterial or chemical products (e. g., sepsis, uremia, etc.). To the latter group belongs the vomiting accompanying migraine.

Vomitus—

1. Mucous, in chronic catarrh of the stomach; after swallowing large quantities of expectoration, in nasopharyngeal and laryngeal inflammation or pertussis.
2. Bilious (yellowish-green or green), in gastroenteric disturbances after repeated vomiting; in peritonitis; intestinal obstruction; liver affections; in the late stages of acidosis.
3. Bloody (hematemesis), in hemophilia and melena neonatorum; congenital obliteration of the bile ducts; cirrhosis of the liver; ulceration of the lining of alimentary tract, especially of the upper part (from corrosive poisons; syphilis, etc.); in vicarious menstruation.
4. Purulent, in rupture into the stomach of large abscesses in the adjacent organs (e. g., empyema).
5. Fecal, in severe intestinal obstruction with reversed peristalsis (e. g., intussusception).
6. Parasitic, in helminthiasis; anchylostomiasis; trichiniasis; echinococcosis.

Diarrhea.*—One to two movements in twenty-four hours are looked upon as normal. But even double the number of evacuations is not necessarily a manifestation of a pathologic condition unless the consistency, color and odor of the stools are materially altered. Since on the first visit a specimen of the stool is not always obtainable, and even if obtained is not invariably of the same consistence as the preceding movements, it is important to gather all the information possible as to the abnormality in question—number, time of occurrence, quantity and quality.

1. Acute diarrhea occurs after the administration of cathartics or corrosives; in indigestion; stomatitis; gastroenterocolitis; proctitis and dysentery (blood, mucus and often pus); acute peritonitis; during the course of divers infectious diseases, especially cholera, typhoid, scarlatina, measles, influenza, sepsis, etc.

2. Chronic diarrhea is observed in dyspepsia; chronic gastroenterocolitis; chronic proctitis and dysentery (amebic); intestinal tuberculosis and other chronic wasting diseases (especially syphilis, leukemia, amyloidosis); helminthiasis (especially in trichocephalus and ankylostomum—often mucosanguinolent stools); malaria (periodic); intestinal lithiasis (mucus, blood and sand), and in partial intestinal stenosis (band-like, flat, mixed with mucus).

Constipation.*—In determining the clinical significance of constipation, inquiry should be made as regards the duration of the constipation, mode of feeding the child, presence or absence of vomiting and tenesmus, and the color and consistency of the stools.

1. Habitual constipation occurs in consequence of insufficient (pyloric stenosis) or improper feeding (excess of fat or starches, etc.); intestinal atony (from a great number of causes, *e. g.*, congenital or acquired muscular insufficiency—megacolon, or artificial distention), general debility, cretinism, etc.; partial intestinal obstruction (*e. g.*, hernia, neoplasms) and abstinence owing to painful lesions in the rectum (*e. g.*, hemorrhoids, fissures).

2. Acute constipation, with persistent vomiting, pain, meteorism, etc., in all forms of congenital intestinal atresia and acquired acute intestinal obstruction (intussusception, strangulation, fecal impaction, peritonitis, appendicitis, and volvulus).

---

*See "Infants' Stools," p. 158; also "Infant Feeding," p. 55.

INFANTS' STOOLS

The character (consistency, color, reaction, odor, etc.) of infants'
stools greatly depends upon the kind and quantity of food consumed.

Normal stools—

1. Soft and pasty, golden yellow, slightly acid and almost odorless,
   in breast-milk feeding.
2. Soft, putty-like, whitish-yellow, slightly alkaline and slightly
   offensive in odor, in cow's milk feeding.
3. Soft, salve-like, yellowish-brown or brown, slightly alkaline
   or neutral, and malt-like in odor, in feeding with malted or
   farinaceous foods.

Abnormal stools—

(a) *Consistency—*

1. Soft, smeary, like moistened shavings of soap, or grayish
   yellow, hard and dry ''soap stools,'' in fat indigestion.
2. Soft or hard and mixed with tough white curds, in casein
   indigestion; hard, lumpy, in habitual constipation.
3. Loose, brown stools mixed with mucus in starch indigestion.
4. Thin, yellowish green in gastroenteritis; typhoid fever;
   from the effects of hydragogue cathartics; rectal stricture
   (*e. g.*, syphilitic).
5. Serous, in severe gastroenterocolitis; cholera.
6. Mucous, in obstinate constipation with tenesmus; in disease
   of the large intestine (colitis, large quantity); in disease
   of the small intestine (mixed with feces).
7. Bloody, in rectal affections (*e. g.*, proctitis, hemorrhoids,
   foreign bodies, fissure, polypus, prolapsus); dysentery;
   intussusception; hemorrhagic diseases (*e. g.*, melena, pur-
   pura, hemophilia).

(b) *Color—*

1. Yellowish-green, in gastrointestinal indigestion (especially
   of casein).
2. Green, in gastroenteritis; excess of sugar; from the effects
   of calomel.
3. Clay-color, in obstruction to the flow of bile.
4. Black, in meconium; from the effects of iron, manganese
   and bismuth; also from blood (coming from upper portion
   of the bowels).
5. Red, from admixture of blood (from lower portion of bowels,
   especially rectum).

(c) *Reaction*—
    1. Decidedly alkaline, in protein indigestion.
    2. Moderately acid, in fat indigestion (from fatty acids); car-
        bohydrate indigestion (acetic or lactic acid).
    3. Strongly acid, in sugar indigestion.

(d) *Odor*—
    1. Foul, in protein indigestion.
    2. Rancid, in fat indigestion.
    3. Sour or pungent, in carbohydrate indigestion.

The stools should be examined also for parasites (see "Intestinal Worms," p. 276) and calculi.

(e) *Bacterial flora*—
    1. In breast-fed feces: *B.* bifidus communis; *B.* acidophilus; few coli; and *B.* lactis aërogenes.
    2. In cow's milk fed feces: *B.* coli communis (splits milk sugar in lactic acid, carbonic acid and water, and partly splits fat in fatty acids) and with it in varying number *B.* acidophilus, micrococcus ovalis; enterococcus (Thiarceli), Gram-staining diplococcus, strepto- and staphylococci, sarcinæ and *B.* lactus aërogenes (splits milk sugar into lactic acid, carbonic acid and water, causing the intestinal contents to become acid).

Principal Abnormalities of Urine

In male infants the urine may be collected by placing the penis in a test tube or the neck of a bottle, fastened by means of strips of adhesive plaster; in female infants, by placing absorbent cotton in front of the vulva, or placing the buttocks on a flat bed pan. Where these measures fail, catheterization should be resorted to.

Traces of albumin and sugar; occasionally hyaline and granular casts; a moderate amount of mucus, uric acid crystals, and urea, are found in the urine of healthy infants in the first few weeks of life.

The quantity of urine passed in twenty-four hours is larger in infants than in older children, but varies with the amount of liquid consumed. It is smaller in breast-fed than in bottle-fed babies.

Polyuria in—
    1. Diabetes mellitus.
    2. Diabetes insipidus.
    3. Contracted kidney.
    4. Granular atrophy of the kidney.
    5. Amyloid kidney.

6. Convalescence after acute diseases (epicritic polyuria).
7. Disease of the nervous system, functional and organic, as hysteria, neurasthenia, migraine, chorea, epilepsy, tabes, cerebrospinal meningitis.
8. Medicinal (acetates, salicylates, digitalis, calomel, etc.).

Oliguria in—

1. Febrile conditions.
2. Profuse diarrhea.
3. Circulatory disturbances.
4. Acute nephritis.
5. Some forms of chronic nephritis.

Anuria in—

1. Atresia urethræ, in the newborn.
2. Uremia.
3. Acute anemia (after severe hemorrhage).
4. Catarrh of the stomach or intestines.
5. Cholera.
6. Dysentery.
7. Nervous manifestations.
8. Lead colic.
9. Poisoning with arsenic, corrosive sublimate, morphine, atropine, oxalic acid, etc.

Glycosuria—

(*a*) *Constant*, in diabetes mellitus.
(*b*) *Transient in*—

1. Cholera.
2. Typhoid fever.
3. Intermittent fever, particularly during convalescence.
4. Syphilis.
5. Scarlatina.
6. Measles.
7. Diphtheria.
8. Influenza.
9. Gout.
10. Disease of the lungs and liver.
11. Disease of the brain, involving the fourth ventricle.
12. Cerebrospinal meningitis.
13. Tetanus.
14. Lesions affecting the central and peripheral nervous system.

15. Poisoning with morphine, atropine, strychnine, oxalic acid,
    carbon monoxide, lead, chromates, chloroform, ether, etc.
(c) *Transient, alimentary in*—
    1. Disorder of the stomach.
    2. Overingestion of starchy and saccharine foods.
    3. Cirrhosis of the liver.
    4. Morbus Basedowii.
    5. Disease of the heart.
    6. Phosphorus poisoning.
    7. Atrophy of the liver.
    8. Traumatic neuroses.
    9. Fatty degeneration of the liver.
    10. Psoriasis.

Acetone in—
    1. Diabetes mellitus, especially in advanced cases; diabetic coma.
    2. Acidosis.
    3. Fever; inanition.
    4. Carcinoma.
    5. Autointoxication.
    6. Psychoses.
    7. After chloroform narcosis.

Diacetic acid in—
    1. Diabetes mellitus, advanced cases.
    2. Autointoxication (diacetonuria) acidosis.

Albuminuria—
    (a) *Renal* (nephritis, pyelitis, pyelonephritis, nephrolithiasis).
    (b) *Vesical* (calculi, colicystitis) ; in tumors.
    (c) *Changes in the constitution of the blood*—
        1. Ischemia.
        2. Anemia.
        3. Struma.
        4. General weakness.
        5. Effect of certain poisons, as cantharides, mustard, oil of tur-
            pentine, carbolic acid, alcohol, lead, etc.
        6. Infectious fevers—Microorganisms in the blood.
        7. Febrile conditions.
    (d) *Disturbance in the circulation*—
        1. Acceleration of the arterial current.
        2. Slowing of the venous current.
        3. Prolonged muscular exercise.

4. After cold baths.

5. After epileptic fits.

6. Compression of the thorax.

7. Derangement of the cerebrospinal system.

(e) *Functional*—
Orthotic, lordotic.

(f) *Digestive*—
Ingestion of excessive quantities of albumin (*e. g.*, eggs, cheese, raw beef).

Casts—

(a) *Hyaline* (narrow and broad), in
Acute and chronic nephritis.

(b) *Granular* (coarse and fine granules), in
Chronic pathologic conditions of the kidney.

(c) *Epithelial*, in
Inflammation in the anatomical structure.

(d) *Bloody*, in
1. Hematuria.
2. Acute diffuse nephritis.
3. Acute renal congestion.
4. Hemorrhagic infarction of the kidney.

(e) *Fatty*, in
Fatty changes in the kidney, large white kidney.

(f) *Waxy*, in
Amyloid kidney and many forms of nephritis.

(g) *Bacterial*, in
Interstitial suppurative nephritis, ascending pyelonephritis.

(h) *Purulent*, in
Abscess of the kidney.

Uric acid (pathologic, when deposit occurs shortly after urine is voided) in—
1. Acute fevers.
2. Increased tissue metabolism.
3. Defective physiologic action of the liver.
4. Sedentary habits of life.
5. Early stages of interstitial nephritis.
6. Convalescence from scarlatina, etc.

Hematuria (blood)—

(a) *Renal*, in
1. Bright's disease.
2. Amyloid disease.

3. Malignant growths.
4. Tuberculosis.
5. Renal calculi.
6. Cystic disease of the kidney.
7. Abscess.
8. Renal embolism.
9. Hydatids.
10. Acute febrile processes.
11. Purpura hemorrhagica.
12. Traumatism involving the kidney.
13. Ingestion of medicines, such as turpentine, cantharides, arsenic, etc.

(b) *Vesical,* in
1. Stone in the bladder.
2. Cystitis.
3. Neoplasms of the bladder.

(c) *Urethral,* in
1. Foreign bodies.
2. Acute gonorrhea.
3. Neoplasms.
4. Traumatism.

Pyuria (pus)—

(a) *Renal,* in
1. Pyelonephritis.
2. Pyelitis.
3. Cancer.
4. Tuberculosis.
5. Nephritic abscess.

(b) *Vesical,* in
1. Cystitis (colicystitis).
2. Vesical stone.
3. Ulceration.
4. Tuberculosis.

(c) *Urethral,* in
1. Gonorrhea.
2. Urethritis.
3. Rupture of abscess in urinary passages.

Peptonuria, in—
1. Croupous pneumonia.
2. Bronchopneumonia.
3. Empyema.
4. Phthisis pulmonum.

5. Epidemic cerebrospinal meningitis.
6. Typhoid fever.
7. Scarlet fever.
8. Malaria.
9. Erysipelas.
10. Purpura hemorrhagica—diverse forms.
11. Scurvy.

Bacteriuria (pathogenic) is the result of infection by the
1. Gonococcus.
2. Tubercle bacillus.
3. Colon bacillus.
4. Strepto- or staphylococcus.

Parasituria—
1. Distomum hematobium.
2. Filiaria.
3. Hooklets of echinococcus.

THE GENITALIA

In the male child we should look for abnormalities of the penis (malformations, adhesions of the prepuce, phimosis, overstretched prepuce from masturbation, faulty location of the urethral orifice, urethral discharge), scrotum and its contents (tumefactions, undescended testicles).

Scrotal tumefactions—
1. Communicating with abdominal cavity, in hernia; hydrocele; and, higher up in the inguinal canal, partly descended testicle.
2. Noncommunicating with abdominal wall, in orchitis (occasionally with parotitis), epididymitis, syphilis, tuberculosis, cysts, and malignant growths of testicle.
3. Dropsical effusions, of renal or cardiac origin or edema from circulatory disturbance in the spermatic cord.
4. Local scrotal inflammation, in abscess, erysipelas, gangrene; sebaceous cysts; traumatism.

In the female we should note the presence of labial hernia or hematoma, vaginal discharge or deposits (in diphtheria and noma); enlarged clitoris or preputial adhesions; atresia vaginæ; abnormalities of the hymen (imperforate).

Vulvovaginal discharge—
1. Mucous, white, in simple catarrhal vulvovaginitis (from lack of cleanliness; irritating urine).

2. Purulent, yellow, or yellowish-green, in gonorrheal vulvovaginitis or infection by other microorganisms (*e. g.,* streptococcus in exanthematous diseases) ; cervicitis.
3. Hemorrhagic, in hemorrhagic diathesis (in the newborn and in older children) ; in vulvovaginitis with erosions of the mucous membrane (sometimes after severe local treatment) ; prolapse of the urethra; neoplasms; menstruatio precox.

THE RECTUM

Abnormalities of the rectum can readily be detected by inspection (sometimes with the aid of proctoscope) and digital examination. We should look for condylomata, fistulæ, prolapsus, hemorrhoids, polyps, prolapse of intussuscepted intestine, fissures, pinworms, foreign bodies and discharges.

Rectal discharges—
1. Mucous, mucopurulent, and slightly bloody, in simple proctitis; rectal fissure or fistula; colitis.
2. Purulent, in communicating ischiorectal abscess; gonorrheal proctitis; impacted foreign body.
3. Hemorrhagic, in hemorrhoids; polyps; dysentery; ulcerative proctitis (tuberculous, or otherwise) ; intussusception; prolapsus recti; hemorrhagic diathesis.

THE VERTEBRAL COLUMN

The vertebral column of the infant under six months is quite straight, except for a slight dorsal curve. As the child grows older and attains the power of sitting, standing and walking, we soon find the dorsal region of the spinal column curving posteriorly and the cervical and lumbar regions anteriorly—compensatory curvatures. At first these curves disappear in the recumbent posture, but they become permanent at about the age of six. The normal spinal column is perfectly movable.

In the physical examination of the spinal column we note the presence of:

Deformities (lordosis, kyphosis and scoliosis)—
1. Congenital, in osteogenesis imperfecta, etc.; cervical rib.
2. Habitual, or postural from faulty posture; the effect of superencumbrance (carrying of heavy weights upon the back or shoulders).
3. Static, the result of oblique pelvis, *e. g.,* congenital or acquired shortening of one lower extremity as in hip-joint disease.

4. Tuberculous, in vertebral caries.
5. Neuromuscular, in muscular insufficiency (to which belongs also rachitic deformity of the spine), or paralysis, *e. g.*, poliomyelitis; pseudoparalysis.
6. Clefts, usually congenital, *e. g.*, spina bifida.

Tumors—

1. Congenital, teratomas; hernial protrusions.
2. Acquired, in vertebral caries, osteoma.

Stiffness* (with or without pain)—

1. Central, in meningitis; meningeal irritation (*e. g.*, apex pneumonia; hydrocephaloid); encephalitis.
2. Spinal, in disease of the spinal cord (*e. g.*, spinal meningitis, myelitis); in trauma or disease of the vertebræ or articulation (*e. g.*, vertebral caries, spondylarthritis). Also cervical rib; osteoma.
3. Neuromuscular, in neuralgia; myalgia; myositis.

THE EXTREMITIES

The extremities should be examined with a great deal of care—inspected, measured, palpated, percussed—since their anomalies in form and disturbances in function, etc., furnish most instructive information not only as to the existence of local disease, but also as to general systemic affections, preeminently those of the nervous system.

Shortness of—

1. Single limbs, in paralytic, hysterical or traumatic (*e. g.*, after fracture) contractures; hip-joint disease; congenital deformities; septic processes.
2. All extremities, in achondroplasia (as compared with the long trunk).

Curvatures—

1. Congenital, in divers congenital malformations (*e. g.*, osteogenesis imperfecta; osteomalacia; achondroplasia).
2. Acquired, after fractures; in syphilis; rachitis; tuberculosis.

Tumefactions—

1. Diaphyseal, tuberculous and nontuberculous, in periostitis; osteitis; osteomyelitis; syphilis; exostosis; malignant growths; after fracture.

*See also "Attitude of the Head and Neck," and "Spondylitis."

2. Epiphyseal, the same as in diaphyseal, also in rachitis; Barlow's disease; arthritis deformans; rheumatic affections; septic arthritides; hemarthrosis (hemophilia, peliosis rheumatica); synovitis; bursitis; "intermittent hydrops."

Muscular Weakness, "flaccidity" (with or without atrophy)—

1. Without true paralysis, in pseudoparalysis of syphilitic origin (upper extremities); Barlow's disease; amyatonia; osteomyelitis; osteomalacia; polyarthritis and myositis; traumatism of the muscles or bones (dislocation or fracture); progressive muscular atrophies (muscular and neurospinal types); idiocy (especially amaurotic family idiocy) and cretinism; rachitis and muscular debility after prolonged sickness (in bed); hysteria.

2. With paresis or paralysis, in poliomyelitis (early); myelitis (the muscular involvement depending upon the seat of the lesion in the cord); Landry's paralysis; spinal meningitis; chronic polyneuritis (usually bilateral and symmetrical) from various causes; birth palsies.

Muscular Contracture, "spasticity" (with or without atrophy)—

1. Without true paralysis, in trismus and tetanus traumaticus and neonatorum; meningismus; early stage of meningitis; tetany; pseudotetany; tetanism $(q. v.,)$; eclampsia infantilis; myotonia (Thomsen); catalepsy; hysteria; trichiniasis; hydrocephaloid.

2. With paresis or paralysis, in all forms of cerebral paralysis (cerebral hemorrhage, embolism, abscess, tumor, sclerosis, tuberculosis, encephalitis, porencephalia, hydro- or microcephalous, etc.); myelitis (late stage); spastic spinal paralysis; amyotrophic lateral sclerosis anterior poliomyelitis (late).

Spasmodic movements*—

1. Intention tremor, in disseminated sclerosis; ataxia hereditaria; spastic cerebral paralysis; myotonia congenita.

2. Irregular shaking, in cerebral hemorrhage; tumor, encephalitis; hydrocephalus; all forms of meningitis; toxic neuritis, especially diphtheritic and uremic; hysteria; Jacksonian epilepsy; idiocy; torsion spasm.

3. Fibrillary twitching, in progressive muscular atrophy; acute febrile diseases; neuroses; strychnine poisoning.

---

*See also "Convulsions," p. 669.

4. Athetoid movements, in chronic brain affections, especially of the internal capsule.

5. Choreiform movements, in all forms of chorea; spasmus nutans; spastic cerebral paralysis; paramyoclonus multiplex; hysteria; tic; lethargic encephalitis.

Paralysis—

(a) *Unilateral*—

1. Upper and lower, in lesions of one cerebral hemisphere, *e. g.*, cerebral hemorrhage, embolism, abscess; tumor, sclerosis, encephalitis, meningitis, depressed fracture, porencephalia, etc.; poliomyelitis.

2. Upper, in unilateral cerebral lesion of the arm center (*e. g.*, embolism, tubercle, etc.); unilateral spinal lesion of the cervical region (*e. g.*, incipient spondylitis, etc.); traumatism to the brachial plexus (*e. g.*, birth palsy); poliomyelitis; regressive stage after hemiplegia.

3. Lower, in unilateral cerebral lesion of the leg center (same as in upper); unilateral spinal lesion in the lumbar region; trauma of the lumbar plexus; poliomyelitis.

(b) *Bilateral*—

1. Upper and lower, in bilateral lesions of the brain (cortex, pons, or medulla), *e. g.*, intracranial hemorrhage, multiple, growths, especially tuberculous and syphilitic, disseminated sclerosis, etc.; spinal sclerosis; spinal meningitis; poliomyelitis; Landry's paralysis (late); progressive muscular atrophy (late); amyotrophic lateral sclerosis (late); syringomyelia (late); multiple neuritis; amaurotic family idiocy (late).

2. Upper, in double trauma of the brachial plexus or individual cords (*e. g.*, compression in instrumental delivery; transverse cervical myelitis); poliomyelitis; Landry's paralysis (early); bilateral cerebral lesions of the arm centers; syringomyelia (early).

3. Lower, in bilateral trauma of the lumbar plexus or its main branches; transverse lumbar myelitis; transverse dorsal myelitis (late); spastic spinal paralysis; hereditary ataxia (late); tabes dorsalis (late); polyneuritis, especially diphtheritic (early); amyotrophic lateral sclerosis (early); poliomyelitis; bilateral cerebral lesions of the leg centers; hydrocephalus.

Localized paralysis of principal muscles concerned in movements of the extremities and their nerve supply—

(*a*) *Upper extremities**—

1. *Trapezius (spinal accessory nerve):* Sinking of shoulder downward and forward; rotation of scapula outward and upward; elevation of shoulder imperfect.

2. *Serratus magnus (long thoracic nerve):* Slight rotation of scapula; difficulty of raising arm above shoulder; deep furrow between scapula and vertebræ on moving arm upward.

3. *Pectorales (anterior thoracic nerve):* Impaired abduction of upper arm; placing of affected hand on healthy shoulder impossible.

4. *Teres major and subscapular (subscapular nerve):* Loss of inward rotation of arm.

5. *Infraspinatus (suprascapular nerve) and teres minor (axillary nerve):* Loss of outward rotation of arm.

6. *Latissimus dorsi (subscapular nerve):* Impaired abduction of arm; inability to place hand on sacrum.

7. *Deltoid (circumflex nerve):* Inability to elevate arm; atrophy.

8. *Biceps and brachialis anticus (musculocutaneous):* Inability to flex forearm, when in supination; inability to supinate forearm, when flexed.

9. *Supinator longus and brevis (musculospiral nerve):* Weakened flexion when forearm is half-pronated; inability to supinate with forearm extended and pronated.

10. *Triceps and the extensors (musculospiral nerve, "radial paralysis"):* Inability to extend forearm (in triceps paralysis); hand-drop in flexed position; flexion of fingers; impaired abduction and adduction (paralysis of the extensors); impaired sensation along radial side; atrophy.

11. *Flexor carpi ulnaris, profundus digitorum, minimi digiti, and inner head of brevis pollicis; the interossei, lumbricalis, palmaris brevis (ulnar nerve, "ulnar paralysis"):* Claw-like deformity of hand.

12. *Pronator radii teres, pronator quadratus, palmaris longus; flexors carpi radialis, sublimis digitorum, profundus digitorum, and longus pollicis (median nerve, "median paralysis"):* Abolition of power of pronation; inability to flex

---

*See also "Birth Palsy," p. 210.

terminal phalanges and thumb; objects can be grasped with the last three fingers only; trophic and sensory disturbance.

(b) *Lower extremities—*

1. *Gluteus maximus and minimus (gluteal nerve):* Difficulty to abduct thigh; to walk uphill; to rise from sitting posture; impairment of circumduction and inward rotation, and walking; toes are turned inward.

2. *Anterior muscles of thigh, except tensor vaginae femoris (anterior crural nerve, "crural paralysis"):* inability to flex thigh on trunk and to flex trunk when in recumbent posture; to extend leg when flexed; difficulty to stand or walk, or to rise from kneeling posture.

3. *Obturator externus and the abductors (obturator nerve):* Impaired adduction and outward rotation of thigh; inability to cross legs.

4. *Biceps, semimembranosus, semitendinosus—the flexors of knee (great sciatic nerve):* Inability to flex knee; difficult locomotion; leg inverted or everted.

5. *Gastrocnemius, soleus, and plantaris—the extensors of the foot (internal popliteal nerve):* Inability to extend (plantar flexion) of foot, to stand on tiptoe; difficulty in walking; foot everted, ankle lowered (talipes calcaneus).

6. *Peroneus longus (musculocutaneous):* Foot inverted; plantar arch flattened (flat foot).

7. *Tibialis anticus, and extensor longus digitorum—flexors of foot (anterior tibial nerve):* Impaired flexion; abduction and adduction (talipes equinus).

8. *Peroneus brevis, and tibialis posticus (posterior tibial nerve):* Inability to adduct or abduct foot without flexion or extension. Talipes valgus in tibial paralysis; talipes varus in peroneal paralysis.

Peculiarities of gait—

1. Dragging, in multiple sclerosis; spastic spinal paralysis; poliomyelitis involving both legs; amyotrophic lateral sclerosis; hemiplegia, and cretinism.

2. Straddling, in tabes dorsalis.

3. Staggering, reeling, in multiple neuritis; hereditary ataxia; cerebellar disease.

4. Waddling, in progressive muscular dystrophy; bilateral dislocation of the hips; rachitis.

5. Hobbling, in osteomalacia.

6. Shuffling, in hysterical paralysis.

Tendon reflexes—

   (*a*) *Knee-jerk*[1]—

      1. Exaggerated, in spinal or cerebral paralysis, associated with "spasticity" of the muscles (see p. 167); also in transverse myelitis affecting the spinal cord above the second lumbar vertebra; cerebellar disease; general nervousness.

      2. Diminished or lost, in spinal or neural affections associated with "flaccidity" of the musculature (see p. 167); also in transverse myelitis below the second or third lumbar vertebra; hereditary ataxia; "meningismus" (early stage).

   (*b*) *Ankle clonus*[2]—

      1. Absent or very slight, in good health.

      2. Present, and often very pronounced, in cerebral hemorrhage; spastic spinal paralysis; dorsal myelitis; disseminated lateral sclerosis; hysterical paralysis; tetanus.

   (*c*) *Periosteal reflex*[3]—

      1. Slight, in good health.

      2. Greatly exaggerated, in cerebral hemorrhage.

Kernig's sign (inability to extend legs when the thighs are flexed on abdomen): In divers forms of meningitis; occasionally in typhoid fever.

Babinski's reflex (extension of great toe with flexion of other toes on crossing sole of foot with index finger): Pathognomonic of meningitis in children over two years of age, in organic hemiplegia.

Brudzinski's sign (flexion of head upon chest produces simultaneous flexion of legs towards abdomen): In meningitis and polioencephalitis.

Weight and Length of Normal Children

An exact record of the gain or loss in weight of the patient is invaluable in the diagnosis, prognosis and treatment. There is no absolute standard for the normal weight or height of a normal infant or older child. To a great extent it depends upon the race the child descends from and also upon the family disposition. Furthermore, the size of the child is not always an indication of its inherent vigor. Ordinarily boys are heavier than girls.

---

[1]Obtained by a sharp blow over ligamentum patellæ, while lower leg hangs loosely down.

[2]Rhythmic oscillation of the foot, elicited by abruptly pressing toes upward with one hand, while supporting the leg with the other hand.

[3]Jerk of hand or forearm produced by a tap upon the tendons of the supinator longus and biceps at lower end of the radius and ulna; or of the triceps tendon, at the olecranon.
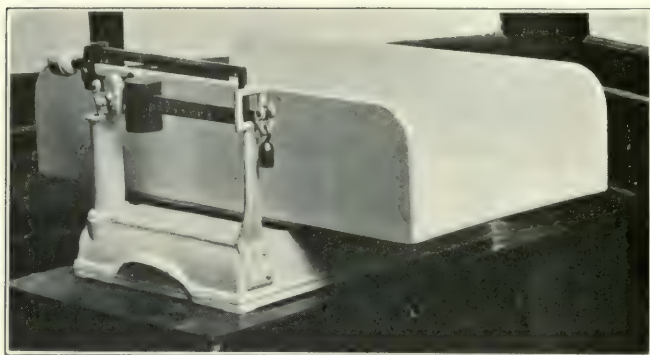
Fig. 29.—Buffalo scale.
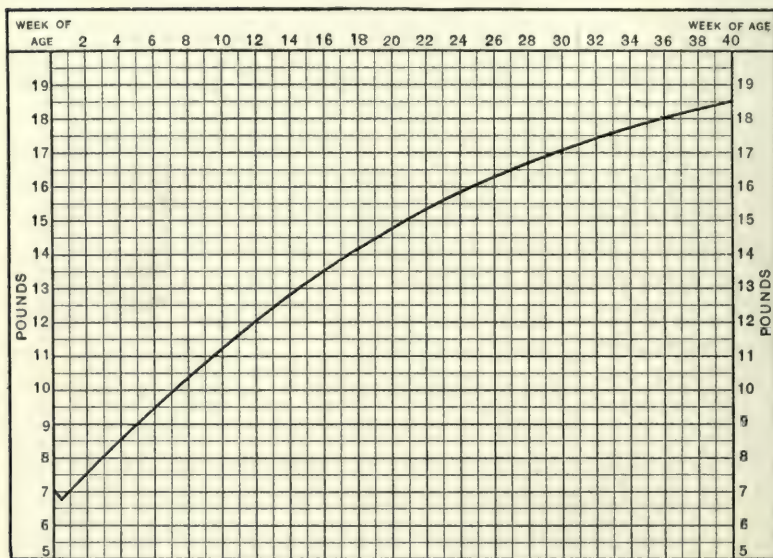
Fig. 30.—Normal infant's weight chart.

Age	Weight Lbs.	Height In.	Circumference of		
			Head In.	Chest In.	Abdomen In.
1 Month	8	21.75	13.75	13.50	13.50
2 Months	10½	23.25	15.40	14.09	14.09
3 Months	12	24.00	15.80	14.70	14.70
4 Months	14	24.75	16.14	15.30	15.30
5 Months	14¾	25.21	16.60	15.88	15.88
6 Months	15½	25.75	17.00	16.07	16.07
7 Months	16¼	26.00	17.16	16.90	16.75
8 Months	17	26.50	17.37	17.00	17.00
9 Months	17¾	26.75	17.50	17.25	17.25
10 Months	18½	27.25	17.66	17.50	17.50
11 Months	19¼	27.75	17.82	17.74	17.75
12 Months	20	29.00	18.00	18.00	18.00
14 Months	21	29.00	18.16	18.16	18.16
16 Months	22¾	29.50	18.33	18.33	18.33
18 Months	23½	30.00	18.50	18.50	18.50
20 Months	24	30.50	18.62	18.62	18.62
22 Months	24½	31.00	18.83	18.83	18.83
24 Months	25	31.50	19.00	19.00	19.00
28 Months	27	33.00	19.16	19.33	19.16
32 Months	29	34.00	19.33	19.66	19.33
36 Months	31	35.00	19.50	20.00	19.50
3½ Years	33	36.50	19.71	20.50	19.71
4 Years	35	38.00	20.00	21.00	20.00
4½ Years	38	38.50	20.21	21.21	20.20
5 Years	41	41.50	20.50	21.50	20.00

Children over five years of age ordinarily gain about 5 pounds in weight and 2 inches in height yearly up to twelve years of age.

Weight is—

1. Diminished, rapidly, in cholera infantum; acute febrile diseases; athrepsia; chronic wasting diseases, especially tuberculosis, malignant growths and suppurative processes; diabetes.
2. Slowly, in dyspepsia; organic affections with slow course, e. g., heart and kidney diseases. (Avoid mistaking increasing weight from large dropsical effusion for natural gain!)
3. Increased, rapidly, in adipositas; pituitary disease (Froehlich's syndrome); cretinism; anasarca.
4. Increased, slowly, in normal health.

Height is—

1. At a standstill or nearly so, in infantilism; cretinism; severe forms of rachitis; achondroplasia; in marked central paralysis.
2. Increased, rapidly, in disease of the hypophysis; acute febrile diseases, especially typhoid fever.
3. Increased, slowly, in normal growth.

CHAPTER III

CONGENITAL MALFORMATIONS

Congenital malformations depend upon the following causal factors:

1. Hereditary disposition (*e. g.*, supernumerary fingers and toes).

2. Antenatal constitutional diseases, especially syphilis and tuberculosis (*e. g.*, hydrocephalus and spina bifida).

3. Traumatism during pregnancy (*e. g.*, multiple fractures and dislocations).

4. Extra- or intraabdominal pressure through pelvic deformities, tumors, etc. (*e. g.*, talipes).

5. Constriction by amniotic bands (*e. g.*, amputations).

CONGENITAL MALFORMATIONS OF THE HEAD

Cephalocele (Hernia of the Brain)

Meningocele, Encephalocele, Encephalocystocele or Hydroencephalocele

Congenital defects in the cranial bones permit the protrusion of a portion of the contents of the skull. The hernia may consist of—

(*a*) Meninges (which form the hernial sac) with or without cerebral fluid—*meningocele.**

(*b*) Meninges and brain substance—*encephalocele.*

(*c*) Meninges and brain substance, which enclose a cavity which is filled with fluid and communicates with a cerebral ventricle—*hydroencephalocele* or *encephalocystocele.*

In accordance with their location we distinguish the following forms of cephalocele:

(*a*) *Cephalocele occipitalis superior*—situated above the external occipital protuberance.

(*b*) *Cephalocele occipitalis inferior*—situated below the protuberance.

(*c*) *Cephalocele nasofrontalis*—emerges from above the nasal bones.

(*d*) *Cephalocele nasoethmoidalis*—situated below one of the nasal bones.

(*e*) *Cephalocele nasoorbitalis*—appears at the inner angle of the eye.

---

*Congenital meningocele is not to be confounded with acquired so-called pseudomeningocele or meningocele spuria s. traumatica, which is either a result of trauma during delivery or a carious process, especially syphilis. Here the tumor is usually situated at one of the parietal bones, increases in size with the development of the brain or enlargement of the cleft in the bone.

The presenting tumor varies in size from a small nut to a fetal head. It may be flat, sessile, hemispherical, pear-shaped or pedunculated. Small tumors are soft and elastic, larger ones pulsate and are often translucent. They enlarge during crying, and may be reduced in size by compression, a procedure which is usually attended by meningeal disturbances. By bearing in mind the characteristic signs, there ought to be no difficulty in differentiating cephaloceles from extracranial cysts, hematomas, abscesses, etc. The diagnosis may be facilitated by an x-ray examination, showing the edges of the opening in the bone. Cephaloceles may remain small and give rise to but very little disturbance. As a rule, however, they grow rapidly and produce death from meningitis, convulsions, or rupture, or proceed a slower course manifested by more or less pronounced backwardness in physical and mental development and other evidences of organic brain disease.

Small cephaloceles require no surgical interference, but merely protection against external injuries by suitable caps, etc., or gentle compression after reposition of the protrusion. Inoperable cases are those complicated by pronounced flattening or diminution in size of the skull, by hydrocephalus or other serious malformations, or where the cleft in the skull reaches down to the foramen magnum. In all other cases removal of the protruding brain is the only proper treatment, followed, if necessary, by osteoplastic closure of the defect in the skull.

As the operation is not rarely successful, if performed by a skillful surgeon; and as the prognosis is extremely grave in large tumors if let alone, there is sufficient justification for early (!) surgical interference.

Hydrocephalus (See pp. 117, 596, 710).

Microcephalus (See p. 707).

CONGENITAL MALFORMATIONS OF THE FACE
Including Those of the Palate, Mouth, Eyes, Nose and Ears

Clefts of the Face and Lips

1. *Median,* the result of nonunion of both globular processes of the central nasal process. This cleft is rarely extensive.

2. *Lateral (labium leporinum, harelip, cheiloschisis),* produced by failure of union of one or both globular processes with the superior maxillary processes. Clefts of the upper lip may accordingly be unilateral or bilateral, may exist as a mere notch into the skin margin of the lip, or, more frequently, extend for some distance upward, involving the whole lip, nostril and upper jaw. It is occasionally associated with cleft palate.

3. *Oblique (meloschisis),* arises from defective closure of the groove between the lateral nasal process and the superior maxillary process. The cleft runs as high as the lower lid.

4. *Transverse (macrostomia),* as a result of patency of the groove between the superior maxillary process and the first branchial arch (mandibula).

Occasionally fistulas and fissures are observed in the bridge of the nose and lower lip.

For details of treatment the reader is referred to text books on surgery.

Fig. 31.—Harelip.

Cleft Palate (Palatum Fissum, Palatoschisis)

It is due to defective union of the processes of the superior maxillary and palate bones which during intrauterine life normally grow inward to meet the vomer in the middle line and the intramaxillary bone in front to form the hard and soft palates.

1. *Complete (Uranoschisma).*—The fissure extends in the middle line through the uvula and the soft and hard palates, and thence through the alveolar process in the line of suture either on one or both sides of the intramaxillary bone. It is generally combined with double or single harelip, and is then designated ''Wolf's Jaw.''

2. *Partial (Uranocoloboma).*—It may involve the uvula only, or part of the soft and hard palates as well. Sometimes it is limited to a mere notching of the alveolar process on one or both sides and forms the continuation of uni- or bilateral harelip.

The consequences of cleft palate, if extensive in degree, are by far more serious than those of cleft lip. Suction and deglutition are greatly interfered with. In older children the voice, articulation, sense of taste, smell, and hearing may all be impaired.

The management of cleft palate is principally surgical. The earlier the operation is undertaken the more perfect are the results. The mode of feeding frequently presents great difficulty. Infants born with marked cleft palate who are unable to nurse have to be fed artificially either with the spoon or through a tube passed through the nose into the stomach. A vulcanized rubber plate covering the defect in the palate often acts admirably.

Defects of the Mouth and Tongue

Atresia Oris (Microstomia).—The lips may be grown together partially or completely. In the latter event an immediate plastic operation is inevitable. Congenital microstomia should not be confounded with the acquired contractures of the oral orifice resulting from syphilis, gangrene, burns, etc.

Adhaesio Linguæ (Ankyloglossia, Tongue-Tie).—It is produced by a large and anteriorly displaced frenulum, and varies greatly in degree, the insertion of the frenulum sometimes extending so far forward as to interfere with suckling, and, later, with speech.

The anomaly may be removed by nicking the frenulum with a scissors, and further "loosening of the tongue-string" with the finger, thus avoiding injury to the ranine artery (dangerous in hemophilia!). The rare adhesion between the epithelial surfaces of the tongue and the floor of the mouth can be liberated in a similar manner.

Macroglossia (Large Tongue).—Enlargement of the tongue may be due to a true lymphangiomatous tumor (cavernous macroglossia), or to a fibrous hypertrophy (fibrous macroglossia). Both forms may coexist. The tongue may be so markedly enlarged as to find no room in the mouth, and by protruding from it become bruised, chapped and cracked, assume such dimensions as to render suckling very difficult or impossible, and possibly lead to a fatal issue from inanition. Congenital macroglossia from the aforementioned causes is not to be mistaken for protrusion of the tongue associated with cretinism. Mild degrees of macroglossia usually improve spontaneously with the growth of the oral cavity; severe forms call for removal of a wedge-shaped piece of the protruding tongue.

Malformations of the Eyes

Anophthalmus (Absence of One or Both Eyes).—This is a rare malformation. In a great many cases careful anatomic examination reveals the presence of rudimentary eyes. If only one eye is absent, the existing eye may be perfectly normal or defective in various ways.

Treatment.—If suitable glasses give the patient sufficient vision for educational and other purposes, an operation may be indefinitely postponed. Otherwise discission, iridectomy or lenticular extraction is indicated.

Coloboma Iridis (Iridoschisma, Fissure of the Iris).—It is usually bilateral and sometimes associated with coloboma of the choroid, fissure of the upper eyelid without involvement of the external skin, microphthalmus, and cataract. If uncomplicated, it disturbs the vision but slightly.

Irideremia (Aniridia).—Partial or complete absence of the iris usually occurs on both sides and is associated with abnormality of the cornea and poor vision. The pupils are iridescent like cat's eyes, and owing to too strong perception of light, the affected children convulsively open and close the eyelids. The same phenomenon is often observed in *albinism*—a condition in which there is a congenital deficiency of pigment in the iris and choroid. Albinos have a blue iris and very fair complexion.

Treatment.—Exclusion of superabundance of light by means of dark glasses or artificial diaphragm.

Malformations of the Nose

Adhesions Between the Turbinated Bones, Particularly the Inferior, and the Septum.—The adhesions may be membranous or bony, and not rarely associated with deflection of the septum. The treatment is the same as in the acquired adhesions.

Atresia of the Posterior Nares.—The closure may be membranous or bony; in the latter condition there is bony union between the palate and the sphenoid. If the closure is only moderately firm, it can be perforated by a stout probe or galvanocautery. Firm bony unions giving rise to difficult suckling call for the employment of chisel and mallet or trephine, using finger in the nasopharynx as a guide to prevent the instrument from penetrating too deeply.

Malformations of the Ears

Fissures and Fistulas of the Ear.—*Fissures* (beneath the tail of the helix) and *fistulas* (in front of and above the tragus) are occasionally observed, especially in connection with other congenital malformations. Deep fistulæ sometimes secrete a serous fluid not rarely causing intractable eczema and requiring operative interference.

Auricular appendages in the form of scattered round or oblong, smooth or warty pieces of cartilage are not rarely found in front of the ear. They can readily be removed by knife or electric cautery.

Ear prominence is a malformation which can often be remedied in the newborn by keeping the ear properly bandaged for several weeks. Sometimes it calls for a slight operation.

Atresia auris, absence of the auditory meatus, is most frequently complete, involving the cartilaginous as well as the bony portion of the canal. Moreover, there is usually also an abnormal tympanic membrane. Hence very little benefit can be expected from operative interference.

All sorts of ear deformities are encountered in connection with idiocy and the allied mental deficiencies (*q. v.*).

MALFORMATIONS OF THE LARYNX AND TRACHEA

Congenital Diaphragm of the Larynx.—The glottis is more or less occluded by a membrane running transversely across the vocal cords. The symptoms stand in direct relation to the size of the remaining opening.

In marked cases the membrane should be excised after preliminary tracheotomy.

Laryngocele and Tracheocele (Aerocele).—The tumor is situated laterally or in the median line. It increases in size on coughing or crying and diminishes on pressure.

The treatment consists of excision of the cyst and closure of the communication with the respiratory tube.

Stridor Congenitus (Child-Crowing).—This congenital anomaly is not to be confounded with laryngospasmus (spasmus glottidis, see p. 677) which is an acquired affection and forms a symptom of spasmophilia (*q. v.*).

The etiology is still indefinite, although in a number of cases the stridor could be traced to malformation of the epiglottis and hypertrophy of the thymus gland.

Stridor congenitus is manifested by a loud, crowing inspiration, accompanied by retraction of the jugulum and epigastrium. It is free from cyanosis or any systemic disturbance, and usually subsides spontaneously in the course of a year or so.

MALFORMATIONS OF THE NECK

Fistula Colli Congenita.—This is a rare anomaly, the result of defective closure of the second and third branchial arches. The fistula is situated either laterally immediately above the sternoclavicular articulation, or medianly at a varying level between the hyoid bone and the jugulum. The fistula becomes apparent by its fine, pinhead-

sized opening with an irregular, moist surface. By passing a fine probe the fistula is found to end either blindly or in the pharynx or esophagus. So long as its track is free, the fistula gives rise to no serious symptoms. Its occlusion, however, is associated with danger of retention of the mucoid secretion and cyst formation. Hence the indication for complete extirpation of the fistulous canal.

Branchial Appendages.—These occur in the shape of warts, nipples or mushrooms, along the margin of the sternomastoid, between the sternoclavicular region and the hyoid bone, consist of skin alone or of skin and cartilage, and are frequently associated with auricular attachments (*q. v.*). They cause no annoyance except from a cosmetic point of view. They are readily removable and nonrecurrent.

Branchiogenetic Cysts.—The seat of these variously sized (from a small nut to a hen's egg), elastic, serous, seromucous, sebaceous, sometimes dermoid cysts is the anterior region of the neck (in the middle line or at the side). The cyst contents may become purulent through infection or sanguinolent through involvement of a blood vessel. Aspiration is a useful aid in the diagnosis, and extirpation of the cyst the only rational mode of treatment.

Hygroma Cysticum Colli Congenitum (Lymphangioma Cysticum).— This tumor consists of a number of small or large communicating or noncommunicating cysts. It varies in size from a slight swelling under the lower jaw or over the clavicle to an enormous tumor embracing the whole neck, and extending downward to the chest and upward to the face. It may even involve the mouth, throat, base of the cranium and mediastinum. In the latter event the prognosis is extremely grave. As the removal of large tumors is attended by great difficulties, it is often justifiable first to try aspiration with subsequent injection of iodine or incision and antiseptic packing. Small hygromas should unhesitatingly be extirpated.

Cervical Rib.—The supernumerary rib is a hard, bony clasp which begins usually at the seventh or sixth cervical vertebra and either ends there as a small protuberance or continues farther to join the first thoracic rib or even the sternum. It may be unilateral or bilateral (Fig. 33). The symptomatology depends upon the degree of pressure exerted by the rib upon the neighboring structures, especially the subclavian artery and some branches of the brachial plexus (neuritis); sometimes there is dilatation of the pupil owing to pressure paralysis of the cervical sympathetic nerve. As a rule, the symptoms do not become manifest until the child has reached the age of eight years or later. The diagnosis must rest chiefly upon a very careful roentgen-ray examination. A nine-year-old girl under my observation was for two years

treated for cervical spondylitis without the slightest benefit, until at last an exact radiogram disclosed the presence of a cervical rib on the right side.

Treatment.—Where the symptoms are mild, palliative therapeutic measures, such as rest, massage and electricity for the relief of pain,
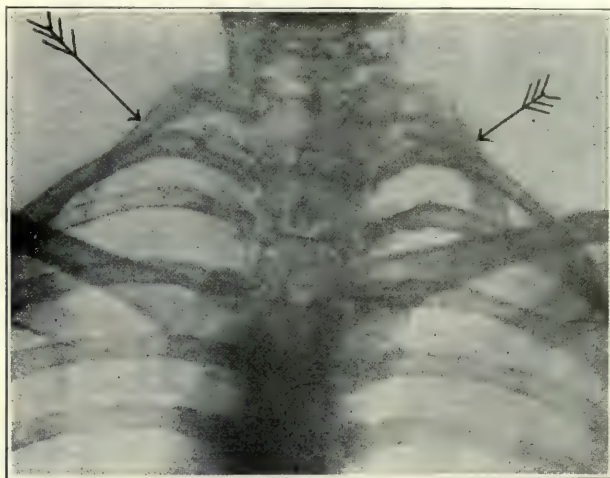
Fig. 33.—Large asymmetrical cervical ribs; neuritis and vascular disturbances in the right arm.   (Dr. A. Church.)

usually suffice. On the other hand, cases presenting severe vascular, nervous and trophic disturbances call for extirpation of the supernumerary rib, an operation demanding great surgical skill.

MALFORMATIONS OF THE THORAX

Defects of the Sternum.—Partial or complete absence or smaller congenital clefts of the sternum are of rare occurrence. They give rise to hernial protrusions of the lung, which if small in size are apt to be mistaken for soft tumors or abscesses. Lung hernia is reducible on pressure, changes in size and shape with respiration, and is frequently associated with paroxysms of coughing.

Among the diverse deformities of the sternum, congenital, nonrachitic "funnel chest" is deserving of special mention. It differs from acquired rachitic funnel-shaped chest by the absence of other rachitic deformities.

Anomalies of the Ribs.—One or more ribs may be absent or rudimentarily developed. The intervening space is filled with membrane.

There may also be accessory ribs (see "Cervical Rib," p. 181, or several ribs may be united.

Defects of the Thoracic Muscles.—Congenital, partial or total absence of one or several of the thoracic muscles is apt to be mistaken for progressive muscular dystrophy. The former, however, is unilateral, while the latter is bilateral. Secondary scoliosis is apt to follow the congenital muscular defects.

All the aforementioned malformations of the thorax require some mechanical contrivance, to prevent either injury to the internal structures or secondary deformities.

MALFORMATIONS OF THE ALIMENTARY TRACT

Atresia Esophagi.—Congenital esophageal strictures are very rare. They give rise to difficulty of swallowing and immediate regurgitation of the food through the mouth and nose. Introduction of a bougie shows the seat of the obstruction.

The treatment is the same as in acquired esophageal strictures. Owing to the absence of true scar tissue in the congenital form, the prospects of recovery are brighter.

Stenosis Pylori Congenita.—(See p. 242.)

Congenital Stenoses and Atresiæ of the Intestines

Any portion of the intestines may be congenitally malformed or completely obliterated. Partial stenosis is most frequently observed in the small intestine, while complete atresia occurred more frequently in the rectum and anus. Pathologically it is found that the lumen of the intestine above the occlusion is widely dilated, while that below it is more or less collapsed.

The symptoms vary with the seat of the lesion. The higher the stenosis, the earlier and more pronounced the vomiting, the larger the quantity of the meconium, and the more marked the dyspnea, and eventually the cyanosis as a result of compression of the thoracic organs by the highly distended stomach.

On the other hand, the lower the stenosis, the more fecal the vomiting, the greater the meteorism, and the more marked the disturbances of the bladder and kidney (partial or total anuria as a result of compression of the ureters by the highly distended intestines). In stenosis of the duodenum the vomitus contains bile substances.

Associated with the local symptoms of intestinal stenosis are: dry tongue, subnormal temperature, rapid emaciation, pinched features of

the face, and collapse. Death usually takes place within a week. Where the stenosis is only partial and slight, the child may linger for months and ultimately recover.

In mild cases the treatment should be symptomatic, principally to relieve constipation and to mitigate the pain and agony. Surgical intervention as a last resort.

Congenital Hypertrophy and Dilatation of the Colon

(MEGACOLON CONGENITUM, HIRSCHSPRUNG'S DISEASE)

This congenital affection should not be mistaken for acquired dilatation of the large bowel associated with intestinal atony from various causes.

Fig. 34.—Moderate degree of megacolon congenitum or Hirschsprung's disease, in a child three years old.

The congenital dilatation is manifested soon after birth by retention of the meconium, although the child is otherwise apparently healthy and free from congenital stenosis of the anus or rectum. Intestinal

irrigation brings forth but a small quantity of feces. The infant is restless and constipated, and its abdomen gradually becomes greatly distended. Some time later the constipation is followed by more or less copious diarrhea due to intestinal irritation from retained feces. After expulsion of the stool and gas, the abdomen is reduced in size, but after a short time it again becomes distended, giving rise to the aforementioned symptoms. Most infants succumb early to the disease, from interference with the thoracic organs or autointoxication by

Fig. 35.—Congenital absence of anus and rectum and of scrotum and its contents.

the decomposing intestinal contents; others may live longer and in rare instances even entirely recover.

Postmortem examination reveals either of the following conditions: (1) Simple dilatation and often lengthening of the colon; (2) ectasis of a section of the colon with or without compensating dilatation or hypertrophy of the adjoining portions; (3) general enlargement of the intestinal lumen and hypertrophy of its walls. The hypertrophy usually involves the longitudinal and circular muscular fibers.

The treatment is chiefly symptomatic (see "Constipation"); in severe cases surgical intervention.

Atresia of the Rectum and Anus

Atresia Ani Proper (Imperforate Anus).—The rectum is normal and ends blindly into the completely closed anus. There may not be the slightest indication of an anus, or the latter is indicated by a few comb-like prominences, a small fossa, or a round indulation.

Atresia Recti.—The anus is normally developed, but the rectum ends blindly somewhere higher up in the canal.

Fig. 36.—Stomach and intestines of case shown in Fig. 35, showing ending of colon in a blind pouch filled with meconium.

Atresia Ani et Intestini Recti.—In this condition the anal orifice is absent and the rectum is arrested in its development higher up, usually in the region of the sacroiliac symphysis.

Atresia Ani Complicata.—There is atresia of the anus, and the rectum terminates either (1) in the bladder (atresia recti vesicalis); (2) in the vagina (atresia recti vaginalis), or somewhere in the urethra (atresia recti urethralis).

Atresia Recti cum Fistula.—The anus proper is occluded; the rectum ends blindly, but is connected with the outer skin by a fistulous

tract. The anal orifice is thus located in an abnormal position in the perineum, vulva, scrotum, etc.

The diagnosis of imperforate anus or rectum usually presents no difficulty. Imperforate anus can readily be made out by inspection. Absence of meconium in the presence of a normal anus indicates that the defect is somewhere higher up. Digital or instrumental examination rarely fails to locate the seat of obstruction. Atresia ani complicata may be detected by the presence of meconium in the urine or by continuous escape of feces from the abnormal communications. The latter symptom is indicative also of atresia recti cum fistula, which can readily be seen.

Imperforate anus and rectum are the only two conditions giving rise to immediate more or less grave symptoms. The child passes no meconium, appears restless, strains, cries, its abdomen is distended, it suffers from dyspnea, and vomits occasionally. If not relieved, it succumbs within a week from rupture of the intestines and peritonitis. Prompt operative interference is therefore imperative. If the obstruction is in the anus, or in the lower part of the rectum, puncture or incision with consecutive dilatation will often suffice to effect a cure. Whenever the point of the atresia cannot be discerned, an artificial anus should be made for quick relief, postponing the curative measures for later. An operation should be postponed also in all other forms of atresia ani or recti, where the escape of meconium is not entirely interfered with.

DEFECTS OF THE ABDOMINAL PARIETES

Diastasis Recti Abdominis.—Lozenge-shaped separation of the abdominal wall, extending from the xiphoid to the umbilicus, is congenital in nature and due to defective closure of the deep layers of

Fig. 37.—Diastasis recti abdominis in an amaurotic idiot.

the abdominal coverings. It is sometimes associated with umbilical hernia.

The symptoms make their appearance when the child is able to run and jump, and consist of sudden attacks of colic (not to be mistaken for enteralgia!), uneasiness in the epigastric region, pallor, etc., which subside when the child is perfectly at rest. These paroxysms are due to partial incarceration of the stomach in the abdominal slit, and should be remedied by bringing and keeping the separated recti muscles together by means of plaster straps or suitable bandage.

Congenital Umbilical Hernia

(HERNIA FUNICULI UMBILICALIS, EXOMPHALOS, OMPHALOCELE CONGENITA, ECTOPIA VISCERUM, AMNION NAVEL)

Fig. 38.—Congenital umbilical hernia.

As a result of faulty development of the abdominal coverings, instead of an umbilicus, a variously sized, sac-like dilatation is occasionally observed which may contain intestinal loops, the stomach, liver, spleen, etc. The hernial sac is composed of the amnion and pari-

Fig. 39.—Congenital femoral hernia.

Fig. 40.—Ectopia viscerum.

Fig. 41.—Thoracoabdominopagus with prolapse of intestines.

etal peritoneum. At birth the contents of the sac can usually be recognized through the thin, transparent membranes, but small protrusions into the cord are apt to be overlooked, and carelessly tied off with the umbilical rest. If there is considerable eventration, the in-

Fig. 42.—Skiagram of thoracoabdominopagus. (Same as Fig. 41.)

fants die early from rupture of the sac and peritonitis. The first indication therefore is to replace the prolapsed structures into the abdominal cavity and to keep them there by means of a suitable bandage. In this manner small hernias not rarely subside spontaneously. Large hernias should be treated by a radical operation.

Persistence of the Ductus Omphalomesentericus

(VITELLOINTESTINAL DUCT)

Physiologically, the omphaloentericus duct, the embryonic tubular communication between the intestinal canal and the germinal vesicle,

disappears at about the eighth week of fetal life. Occasionally the duct is not obliterated, and leads to the following principal abnormalities:

1. A fine fistula at the umbilical ring, forming a communication between the bowels and the exterior, and secreting a cloudy fluid containing a trace of fecal matter.

2. A hernial protrusion through the umbilicus in the form of a red finger-shaped tumor which is usually composed of the prolapsed walls of the fistula, but sometimes of intestinal loops.

3. Open Meckel's diverticulum. It is a blind appendage of the lower part of the ileum, and may be free or united with the umbilicus by a solid cord. Under certain conditions it may enter a hernial sac and here become strangulated. It may produce "ileus" by incarcerating some loops of the intestines, and give rise to local intestinal inflammation closely resembling that of appendicitis.

Persistent omphaloenteric duct may be mistaken for: (1) persistent urachus—on examination with the catheter it can be reached through the bladder; the secretion is composed chiefly of urine; (2) sarcomphalos—has no fistular opening.

Fine fistulæ frequently close after repeated cauterization with the caustic stick. Wherever the prolapse is very marked, or in cases associated with open diverticula, a radical operation is imperative, since their presence is always a menace to life.

Urachus Fistula

(FISSURA VESICÆ UMBILICALIS)

Persistent urachus—the duct through which the urinary bladder communicates with the allantois—gives rise to a fistulous tract which ends at the umbilicus. On pressure a small hernial tumor arches forward and secretes a clear or turbid fluid, composed of urine alone, or urine, mucus and pus. If the fistula is large, the flow may be continuous. It may give rise to cystitis and even pyelonephritis compelling early operative procedures. The first attempt at a cure should be directed to making the natural outlet free (e. g., cure of phimosis). Small fistulæ often yield to cauterization and continued pressure with a bandage. If this fails, the walls of the sinus should be freshened and sutured.

Its differentiation from persistent ductus omphalomesentericus has been emphasized above.

MALFORMATIONS OF THE GENITOURINARY ORGANS
Congenital Abnormalities of the Kidneys

The kidneys, like all other parts of the body, are subject to defective embryonic development. They may be abnormal in size, shape (horseshoe), and number. This is of clinical importance, since malformed kidneys are more easily affected by disease, especially tuberculosis, than normal organs. *Congenital absence* of one kidney has been observed once in about 4,000 autopsies. Furthermore, it is usually found that whenever one kidney is absent, the other one is in a more or less diseased condition, chiefly greatly hypertrophied. *Congenital displacement* of the kidney (both kidneys on one side; in front of the vertebral column; low down in the pelvis) is very apt to cause many diagnostic errors.

Malformations of the Ureters

Abnormal ureteral openings, as to size and position, are of great clinical significance. In the male the ureter may terminate into the sphincter of the bladder, the prostatic portion of the urethra, or in the seminal vesicles, and by interference with the flow of urine give rise to dilatation of the ureter and renal pelvis and atrophy of the renal parenchyma. In the female the ureter may end in the sphincter of the bladder, in the urethra, or in the vagina. More serious than misplacement is *absence* or *atresia* of the ureter. Either one of these latter conditions invariably produces hydronephrosis, compelling extirpation of the affected kidney. Double ureter, if free from any other anomaly, is not attended by any pathologic phenomena.

Malformations of the Bladder

Ectopia Vesicæ Congenita, Cleft Bladder, Fissure of the Bladder, Extrophy Vesicæ.—Cleft bladder arises from arrest of development of the anterior walls of the bladder and abdomen, and often also of the symphysis. It may be partial or complete. In the complete variety the posterior vesical wall protrudes as a round, moist, bright-red tumor, through a gap in the abdominal wall, situated in the median line between the umbilicus and the urethra. The mass is marked by two small tubercles on both sides—the orifices of the urethra—from which the urine dribbles continuously. In the male this is associated with epispadias of the rudimentary penis; in the female the clitoris is clefted, the labia are widely separated, and the urethra and vagina more or less defective. Eversion of the bladder is often complicated also by other malformations of the body, and in the majority of

instances leads to early death. Partial ectopia vesicæ offers a more favorable prognosis, particularly if a plastic operation is resorted to early. Temporary relief may be obtained from a suitable urinal held in place by means of a truss.

Malformations of the Urethra, Prepuce, Testicles, and Vagina

Atresia Urethræ.—*Total atresia urethræ* is a rare malformation. When it does occur, it is usually epithelial in nature or at most membranous. In the former instance the atresia promptly yields to pressure with the tip of a sound; in the latter, to a small incision and dilatation by means of a small, blunt silver probe.

Complete absence of the urethra is extraordinarily rare.

Congenital stenoses are not rarely found along the urethra, and if presenting no distinct hindrance to urination are frequently overlooked.

In cases of marked urethral stenosis, the still patent urachus often permits the escape of urine through its fistulous tract running from the bladder to the umbilicus.

Misplacement of the Urethral Opening (Epispadias, Hypospadias).— The urethral opening may be situated on the upper part of the penis (epispadias) or at its inferior aspect (hypospadias). The latter abnormality is more frequent than the former. Both conditions are productive of more or less disturbance of urination (incontinence in epispadias, dysuria in hypospadias), secondary intertrigo, erosion and ulceration of the genitalia from the effects of the irritating urine, and later in life interference with virility.

Pronounced hypospadias (perineoscrotal) closely resembles hermaphroditism, and, when associated with retention of the testicles, it may be impossible to determine the sex of the infant.

Except in the very mildest cases early operative interference is indispensable.

Congenital Phimosis

A moderate degree of adherence of the prepuce to the glans penis is physiologic in the newborn. Ordinarily the adhesions disappear spontaneously in the course of time. In some cases, however, the prepuce remains adherent and stenosed at its orifice so that the glans cannot pass through. In consequence there is more or less retention of urine between glans and prepuce (particularly if the latter is elongated or hypertrophied), infection and decomposition of the sebaceous secre-

tion (smegma) and secondary inflammation of the penis and adjacent structures.

In the presence of inflammation urination is difficult and very painful, the infant cries, presses and strains (in predisposed children often the cause of hydrocele, hernias and prolapsus recti), or, fearing pain, retains the urine for many hours, a habit which is apt to give rise to cystitis, pyelitis, and even uremic convulsions.

Phimosis frequently forms also the cause of enuresis, priapism, masturbation, and a number of more or less reflex nervous phenomena.

In mild cases of phimosis the prepuce should frequently be pushed back and forth and the retained smegma removed. When the adhesions are very firm they may be broken up with the aid of a dull probe and kept loose by daily retraction of the foreskin and application of an antiseptic cooling lotion such as lead water or a 2 per cent solution of aluminum acetotartrate. In this manner good results are obtained within a few days.

When the preputial stenosis is the predominating trouble, slight nicking of the preputial ring with scissors (laterally, above, and below), followed, as before, by loosening of the adhesions, daily preputial retraction and local antiphlogosis, is all that will be necessary to effect a permanent cure. This procedure is at all times preferable to circumcision, except in cases of phimosis associated with elongated or greatly hypertrophied foreskin and severe inflammation.

Circumcision, when indicated, should be performed under very careful aseptic precautions, preferably under general anesthesia. The surgeon grasps the prepuce between the thumb and index finger, exerting sufficient traction to draw it from the glans penis, puts over it a shield or forceps just in front of the glans, and with scissors or knife removes the distal, superfluous portion of the prepuce. He next seizes the inner layer of the prepuce, which still covers the glans, with a thumb forceps and with the aid of scissors cuts it so far backward as to enable him fully to expose the glans and bring the edges of both preputial layers in apposition by a fine continuous suture. The dressing should consist of sterile gauze (not medicated! danger of intoxication). Numerous accidents have been reported as the result of circumcision, but all, except uncontrollable hemorrhage in the hemophilic, are preventable. In such hemorrhage the actual cautery should be resorted to without delay and use all other therapeutic measures as recommended for melena (p. 230) and hemophilia (p. 553). Milder hemorrhages will often yield to firm compression of the penis with a hard catheter in the urethral canal.

Cryptorchidism

(UNDESCENDED TESTICLE)

Normally the testicles descend into the scrotum by the end of fetal life. In the event of arrested development or malformation of the canal of Nuck, of a constriction of the inguinal ring, and malformation of the testis, epididymis, or the vas deferens, etc., one (monorchidism) or both (cryptorchidism) testicles are not infrequently retained in the abdominal cavity, at the inguinal ring, or at the upper portion of the scrotum. More rarely the testicles become displaced, and through a false passage emerge either at the crural arch (crural testicle; under the fold of skin between the thigh and scrotum (scrotofemoral testicle); or behind the scrotum (perineal testicle).

In the majority of instances an undescended testicle is free from any serious consequences, and reaches its normal position spontaneously within the first few years of life. Occasionally, however, it may become impacted at the inguinal canal, giving rise to excruciating pain and inflammatory symptoms; if associated with hernia, strangulation may take place in both structures at the same time; it may cause atrophy in the genitalia; it may be the seat of malignant degeneration, and finally, it may be productive of a number of reflex phenomena (epilepsy?).

Cryptorchidism should not be confounded with *anorchidism* or absence from the body of both testicles (this is usually associated with rudimentary penis and, later, absence of spermatic secretion), or with ascent of the testicles from contraction of the scrotum (they descend with relaxation of the scrotum).

Expectant plan of treatment is followed up to puberty in the absence of complications. A capsular truss should be worn in cases of misplacement. Gentle massage is useful. Orchidopexy and other surgical procedures should be instituted as indications arise. Speedy operation in case of strangulation.

Hydrocele

It is a common affection of early infancy and most frequently congenital in nature. Varying with the seat of the accumulation of the abnormal quantity of serous fluid, we distinguish the following kinds:

1. *Hydrocele Tunicæ Vaginalis.*—This is a unilateral, oval, smooth, translucent, more or less tense, fluctuating swelling, which appears first at the lower part of the scrotum, and gradually rises up to the abdominal ring. Posteriorly to the hydrocele usually lies the testicle.

2. *Hydrocele Funicili Spermatici (Hydrocele of the Cord)*.—This form resembles the former; except that the testicle usually lies at the bottom of the scrotum and is distinctly separated from the hydrocele by a constriction. It is sometimes made up of several small cysts simulating a string of beads.

3. *Hydrocele Vaginalis Communicans ("Congenital Hydrocele")*.—This form occurs when the tunica vaginalis preserves its communication with the abdominal cavity and becomes filled with serum, form-

Fig. 43.—Congenital hydrocele communicans.

ing a cylindrical tumor, extending to and through the abdominal ring. It is often associated with hernia (hydrocele hernialis). As the contents of both are reducible on pressure the differential diagnosis between congenital hernia and hydrocele vaginalis communicans is sometimes difficult. In hydrocele, however, the return of fluid to the peritoneal cavity occurs without intestinal gurgling—the reverse being the case in congenital hernia.

Hydrocele often disappears spontaneously, especially after removal of reflex irritation, *e. g., phimosis.* If it persists, we employ local counterirritation (painting with tincture of iodine or mercury ointment), or aspiration, if the hydrocele enlarges. The latter procedure may be followed by the injection of a few drops of tincture of iodine or carbolic acid and alcohol. Absorption of the fluid is hastened by a few large doses of potassium iodide. In hydrocele communicans a truss should be worn to prevent hernia. The pressure exerted will often obliterate the inguinal portion of the vaginal process, and also cure the hernia, if present.

If the aforementioned palliative and curative measures fail—which is rarely the case—a radical operation becomes necessary.

Atresia Vulvæ.—Atresia vulvæ consists chiefly of a cellular adhesion of the labia minora, and may be partial or complete. In total atresia vulvæ there is anuria, with its secondary symptoms, necessitating immediate attention, *i. e.,* forcible separation of the labia with the fingers or with the aid of a dull probe or scalpel. In partial atresia separation of the labia occurs spontaneously.

Atresia Vaginæ Hymenalis (Imperforate Hymen).—This congenital malformation usually escapes observation until puberty, when partial or total retention of the menstrual flow gives rise to local and general disturbances.

Incision and packing with iodoform gauze readily remedies the trouble.

Atresia Vaginæ.—Like the aforementioned malformation, narrowing or complete closure of the vagina is not detected till after puberty. Total atresia vaginæ is usually associated with *absence of the uterus.* This should always be borne in mind before resorting to operative procedures for the relief of the atresia.

CONGENITAL MALFORMATIONS OF THE VERTEBRAL COLUMN

(INCLUDING THOSE OF THE SACRUM AND COCCYX)

Spina Bifida or Hernia of the Cord

Meningocele Spinalis, Myelocystocele, Myelomeningocele.—Analogous to hernia of the brain (see ''Cephalocele''), that of the cord also is divisible in three principal groups: Meningocele spinalis, myelocystocele, and myelomeningocele.

(*a*) *Meningocele Spinalis.*—Meningocele spinalis is a protrusion of the pia mater without participation of the spinal cord. It is filled with cerebrospinal fluid, translucent, often pedunculated and may reach the size of a child's head. It is covered by normal skin. Paralysis is rare. Pressure on the tumor produces bulging of the fontanelles and spasms.

(*b*) *Myelocystocele.*—Myelocystocele is situated on a broad base and is readily replaceable on pressure. The covering skin is greatly distended but normal in color. Palpation reveals that the tumor consists of solid masses in addition to fluid. It is frequently associated with hydrocephalus and accompanied by motor and sensory disturbances.

(*c*) *Myelomeningocele.*—Myelomeningocele is a pear-shaped or spherical fluctuating, tense, broad or pedunculated tumor the size of a walnut to that of a child's head. Its covering skin is bluish, very thin and traversed by numerous blood vessels. It is composed of cord substance and its membranes, and forms a true hernial protrusion through a cleft in the vertebral column. The cleft and to some extent also the hernial orifice can often be felt at the base of the tumor. Myelomeningocele is the most frequent variety of spina bifida and gives rise to marked motor and sensory paralyses.
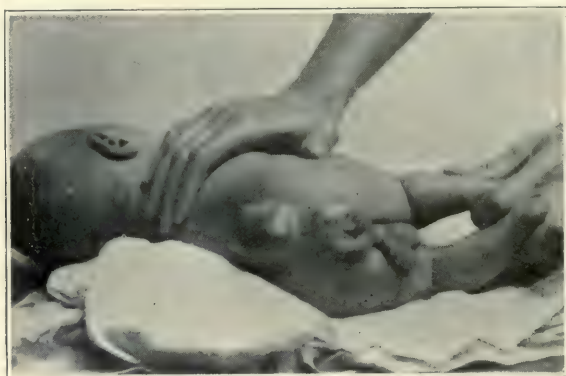
Fig. 44.—Myelocystocele. Note funnel-shaped eversion of the rectum owing to paralysis of the levator and sphincter ani.

Almost all forms of spina bifida are associated with hypertrichosis of the surrounding skin. This is especially pronounced, and indeed, often forming the only outward sign of deformity, in *spina bifida occulta* (a meningocele usually at the sacrolumbar region hidden under masses of fat). The hair is usually so arranged as to form a crown over the center of the defect. When well developed it may resemble a tail. Apart from the malformation the condition of most children at first is perfectly normal. As the tumor enlarges the results of the pressure on the cord or the cauda equina gradually appear. The symptoms vary with the degree of involvement of the spinal cord; they are, therefore, most pronounced in myelomeningocele sacrolumbalis. Here we have motor and sensory paralyses of the legs, of the rectum, bladder, and the perineal

muscles, convulsions and trophic disturbances. In less severe cases, the paralysis may be limited to the legs only. Several years ago (Med. Rec., New York, Jan. 6, 1912) I called attention to persistent incontinence of urine forming a characteristic symptom of spina bifida occulta.

Bearing in mind the characteristic symptomatology of spina bifida, *i. e.*, a more or less translucent, compressible, barely movable, thinly

Fig. 45.—Spina bifida occulta in a boy eight years old. This condition was associated with incontinence of urine.

covered tumor, in the majority of instances associated with paralyses, there ought to be no difficulty in differentiating it from sacrolumbar neoplasms. In cases of doubt the diagnosis may often be cleared up by exploratory puncture and radiographic examination (the latter showing a vertebral cleft).

Spina bifida may sometimes escape notice when it is surrounded by a solid tumor.

The majority of children with marked spina bifida die when very

young, often during birth, owing to rupture of the tumor and shock following rapid escape of the cerebrospinal fluid. Most of those who survive succumb later from rupture of the sac and subsequent infection and purulent meningitis; from gangrene and ulceration of the skin with subsequent sepsis; and finally, from intercurrent diseases and marasmus. Simple meningocele gives the best prognosis if recognized early and protected from external insults by a suitable pad or apparatus.

This palliative method of treatment should always be tried in cases of spina bifida which project very slightly and are covered by normal, well-nourished skin. Aspiration of the hernial sac is useful to relieve the symptoms of compression and to lessen the danger of spontaneous rupture. Aspiration may be followed by injection of iodine or preferably iodine-gelatin. In selected cases it may prove of permanent benefit.

A radical operation is the ideal procedure in suitable cases. However, extensive paralyses, severe irreparable malformations elsewhere, hydrocephalus, and grave systemic affections are contraindications to operation. In such cases palliative and symptomatic methods of treatment are indicated.

Congenital Sacral Tumors

Closely related to and frequently associated with spina bifida (*q. v.*) are congenital sacrococcygeal tumors. They may be classified as follows:

1. *Double Formations*—
   (*a*) *Complete*—two fully formed individuals grown together at the buttocks.
   (*b*) *Incomplete or parasitic formations*—one or several rudimentary portions of the body attached to the buttocks of a fully formed individual.
2. *Sacral Hygromas.*—Single or multiple cysts, attached by a broad base to the dorsal surface of the sacrum. They are sometimes associated with spinal hernia.
3. *Tumores Coccygei.*—Neoplasms attached to the anterior surface of the sacrum and coccyx. The tumors are composed of fibrous or granular masses generally of sarcomatous nature, sometimes of fat, cartilage, or bone. Occasionally they involve the spinal canal, or surround a spinal dural protrusion (spina bifida). They never extend above the lower border of the gluteus, but spread toward the pelvis and between the legs of the child.
4. *Caudal Formations*—
   (*a*) *Complete tails,* manifested by an actual increase in the number of coccygeal vertebræ.

(*b*) *Imperfect tails,* enlargement of vertebral column by rudimentary tissue.

But few children born with coccygeal tumors live beyond the age of one year. As the tumors enlarge, the infants succumb to progressive cachexia and exhaustion.

As a rule, sacral tumors do not interfere with the life of the child if suitable protection is furnished against vulnerability of the tumor and secondary infection. In some selected cases (see "Spina Bifida") perfect results are often obtained by skillful surgical measures.

MALFORMATIONS OF THE EXTREMITIES AND HIP

Of the numerous malformations of the extremities (*e. g.,* complete absence; spontaneous partial amputations; fractures; supernumerary fingers and toes, etc.) but few are of interest to general practitioners—namely, congenital dislocation of the hip and club foot. As these abnormalities are apt to be confounded with similar acquired affections, they will receive special consideration.

Luxatio Coxæ Congenita

(Congenital Dislocation of the Hip)

The dislocation may be unilateral or bilateral. The acetabulum is rudimentary in form, and the head of the femur rests either above it, above and to the outer side, or above and behind it upon the ilium, sometimes immediately at the side of the great sciatic notch. If one leg is displaced it is shorter than the other, giving rise to distinct limping. If both sides are affected the gait is wobbling—"duck gait." As a result of this anomaly the buttocks project prominently backward while the spine is either thrown forward (lordosis, in bilateral) or tilted sideways (scoliosis, in unilateral dislocation). The differential diagnosis between this condition and rachitis and coxa vara is best established with the aid of the x-rays which show the abnormal position of the head of the femur. If the malformation is detected early, (in a certain number of cases the dislocation is acquired as a result of septic arthritis in the newborn) it may be corrected either by opening the joint, replacement and fixation of the head of the femur in the artificially deepened acetabulum, or by bloodless forcible reduction of the deformity and fixation of the head of the femur in the acetabulum by prolonged use of plaster-of-Paris bandages (Lorenz's operation). For details of treatment the reader is referred to textbooks on orthopedic surgery.

Talipes

(CLUB FOOT)

1. *Talipes varus,* inversion of the foot, so that its sole faces the other foot. This is the most common of the congenital forms.

2. *Talipes valgus,* flat-foot, effacement of the arch.

Fig. 46.—Bilateral club feet in father and three children. (After Joachimsthal.)

3. *Talipes equinus,* lowering of the anterior part of the foot, the child steps on his toes.

4. *Talipes calcaneus,* elevation of anterior part of the foot, heel alone touching the ground.

Compound forms may be produced by combination of the different varieties.

The diagnosis of the type of club foot can readily be made by inspection; it is sometimes difficult, however, to differentiate the congenital from the acquired forms, *e. g.,* rachitic or paralytic club foot. In rickets

the distortion of the feet is generally associated with other pathogno-monic symptoms of rickets and is gradual in development. In paralytic club foot (*e. g.*, poliomyelitis) the limb is wasted, flabby and cold and there is a history of postnatal, gradual appearance often in association with other paralytic deformities.

Congenital club foot is being attributed to various causes, but is prob-ably due to some mechanical interference with the normal development of the joints, ligaments or tendon insertions.

Fig. 47.—Same case as Fig. 44 showing also congenital club foot.

Treatment of Club Foot by General Practitioner.—Oettingen re-marks that during the 2,000 years of experiences with club foot, the important part played by the knee in the correction has been almost entirely overlooked. The deformity develops with the knee flexed to the utmost, and it should be flexed at a right angle in the immobiliza-tion. This has the great advantage that the sole can be held in proper position by traction on the knee. Treatment should commence the moment the child is first presented to the physician, even if it is only one day old. No anesthesia is required, merely a stout twilled cotton flannel bandage about 4 or 5 cm. wide. The bandage is passed first around the foot, after the foot and thigh, just above the knee, have been smeared with a soft solution of mastic which glues the fuzzy stuff firmly to the skin. The bandage starts at the little toe and is wound around the foot and then passed from the little toe over the knee and is then brought around and across the front of the leg to the inner aspect of the foot, forming thus a figure-of-eight bandage. Be-

fore applying the bandage the physician should manipulate the foot to bring it with maximal outward rotation of the leg into its normal position, watching the clock to see that he does not take less than five minutes to accomplish this. During the application of the bandage the assistant holds the thigh with one hand and the middle toe of the foot with the other, not releasing it until bandaging is completed. By this means the sole of the foot is held in correct position supported by the thigh, the bandage holding the foot in pronation, outward rotation, abduction and dorsal flexion. It is impossible for the foot to slide back into its old position. This bandage leaves free the entire thigh, the under part of the knee, and the entire inward aspect of the leg and ankle, with a certain possibility for movement in all the joints. These are all immense advantages. The child is allowed to go home for two days, and can be bathed if the leg is held up out of the water. After two days the physician removes the bandage, washes the leg with warm water and soap, and massages the leg for a few minutes. A bandage is then applied, which is left for five days, and is then removed for an interval of four days. In the same way three more bandages are applied, each for a week. By the end of the fourth week the foot is in normal position, ready for the after-treatment. So far, the mother is not allowed to massage or work on the foot, but now she is taught to massage three or four times a day, seizing the leg above the malleolus, the sole toward her as she sits or stands in front of the reclining child. With the middle finger of the other hand on the little toe of the foot, she strokes the sole away from her. This one simple movement combines the four compensating elements for club foot, continuing the pronation, extension, abduction, and outward rotation of both foot and leg. With older children a rubber strap is applied afterward, passing around the foot and knee like the other, with a buckle to keep it fast, tied with tape to keep if from slipping off the knee. Complicated and neglected cases of club foot, of course, require surgical therapeutic measures.

CONGENITAL AFFECTIONS OF THE MUSCLES AND BONES

Amyatonia Congenita

(Myatonia Congenita, Oppenheim)

Amyatonia congenita is characterized by general flaccidity of the muscles, especially of the lower extremities and in a slighter degree of the arms. The neck, cranial nerves and diaphragm are usually normal. Intelligence is occasionally deficient. There is no atrophy, but the patellar reflexes are either diminished or lost. As the affection seems to be due to delayed development of the musculature, it

generally improves, particularly if assisted by massage, baths and electricity, and general tonic treatment. In a case reported by J. B. Holmes* postmortem examination revealed a relatively large spinal cord with the anterior roots diminished in size, as compared with the posterior roots.

Myotonia Congenita

(THOMSEN'S DISEASE)

This is a rare, probably hereditary, affection of the muscular system, characterized by sudden spasm and rigidity of individual or groups of muscles, especially when the patient begins a voluntary movement, e. g., arising from a certain posture, clasping hands, etc.

Fig. 48.—Osteogenesis Imperfecta. Every long bone of the body was repeatedly fractured. The child finally died at the age of two and a half years from cerebral hemorrhage resulting from a slight fall upon the head. (Courtesy of Dr. J. L. Rubinstein.)

Similar tonic contractions occur from the effects of a blow upon a muscle; and the application of a strong (20 to 25 milliamperes) galvanic current produces certain wave-like muscle contractions which move from the area of the cathode to that of the anode. Although often appearing in early infancy, the disease does not endanger life or health. Warm baths and massage may prove of benefit.

Osteogenesis Imperfecta

(FRAGILITAS OSSIUM IDIOPATHICA)

This rare, congenital bone affection of obscure origin is characterized anatomically by the unusual persistence of the interstitial cartilagi-

*Am. Jour. Dis. Child., November, 1920.

nous substance and great deficiency of osseous elements and lime salts in the primary zone of calcification. It involves all the bones of the body. They are soft and thin and readily bend and break on the slightest manipulation; hence the frequency of several fractures during delivery of the baby. Those who survive early infancy, often succumb to fractures of the head, spine or ribs when they begin to stand or walk. Cases reaching adolescence, however, are on record. As fractures of the long bones are not rarely met with in congenital syphilis, osteogenesis imperfecta is apt to be mistaken for the former disease. In syphilis, however, we have several other characteristic signs, including positive Wassermann reaction, which are absent in osteogenesis imperfecta. For its differentiation from rachitis, see p. 511.

Treatment.—Avoidance of traumatism and attention to the general health of the baby. Phosphorus preparations and cod liver oil may be tried, especially in mild cases. As the thymus is not rarely found atrophied, thymus treatment is worth trying.

Achondroplasia (see p. 512).

CHAPTER IV

INJURIES AND DISEASES OF THE NEWBORN

I. BIRTH INJURIES

Nature in its infinite wisdom provides a more or less large quantity of liquor amnii to protect the fetus in utero against undue pressure and possible injury. If, perchance, the amniotic fluid escapes prematurely, either spontaneously or artificially, the fetus, in its descent through the parturient canal, subjected to powerful pressure by the maternal structures or mechanical manipulations, sustains a number of injuries which vary in severity from simple external bruising to grave compound fractures and internal, sometimes fatal, injuries.

A. Superficial Structures
Caput Succedaneum

Vertex presentation being the most common form of delivery, the head consequently stands the brunt of the injuries. The so-called caput succedaneum is a circumscribed edema of the scalp and consists of a serous or hemorrhagic extravasation into the subcutaneous tissues of the scalp. It is observed immediately after birth as a doughy, evenly distributed, variously sized, soft tumor which disappears spontaneously by absorption, unless infected through external abrasions. In the latter event it requires surgical treatment, such as antiseptic dressings, incision and drainage.

Cephalhematoma

More serious than the aforementioned condition is hemorrhage occurring between the pericranium and cranial bones in the form of a circumscribed, elastic, distinctly fluctuating, painless tumor, situated upon the right or left side of the head (sometimes both sides are affected). The cephalhematoma develops gradually within the first few days of extrauterine life, and owing to the firm attachment of the periosteum to the edges of the cranial bones along the sutures, it never extends beyond the latter or over the fontanelle. All around the tumor a hard, bony ridge is soon (after about two weeks) detected, which with the depressed center gives a sensation somewhat like that of a depressed fracture.

Cephalhematoma may be mistaken for caput succedaneum, which appears immediately postpartum and disappears after a day or two; for subaponeurotic or subcutaneous hemorrhages, which occur sometimes also from intrapartum pressure, but extend beyond the sutures; for congenital encephalocele, which lies between but not over the bones, pulsates, enlarges. on crying or coughing, and can be partially reduced; and, finally, for vascular tumors, which are compressible and free from a bony ridge.

The tumor usually disappears spontaneously, sometimes requiring weeks and months to do so. If suppuration occurs, it calls for surgical interference.

Hematoma Sternocleidomastoidei

Pathologically akin to cephalhematoma is the intrapartum hemorrhage which takes place within the sheath of the sternocleidomastoid muscle, as a result of rupture of several muscle fibers and consecutive myositis.

The tumor in the neck is generally observed a few weeks after birth, more rarely earlier, by noting the baby holding its head on the side. It varies in size from that of a hickory nut to a walnut. It is at first soft, later hard and cartilaginous in consistence. Severe hemorrhages may give rise to torticollis.

This condition demands perfect rest to the head, cold compresses for the relief of pain, and later gentle massage to promote absorption of the tumor.

R.
Ung. kalii iod.   (U. S. P.)
Adipis lanae   āā ℥ ii | 8.0
M. ft. Ung.
S.: To be applied with gentle massage once a day.

2. Deep Structures

Birth traumatism is not always limited to the skin and muscles. Now and then the viscera (the lungs, liver, peritoneum, etc.), the bones, the peripheral nerves, the meninges and brain are involved. Fractures and dislocations are not rarely observed, especially in the long tubular bones and the clavicle, while the cranial bones are often badly displaced (the occipital and frontal are pushed under the parietals), fissured (see "Meningocele,"), compressed and fractured, giving rise to grave, frequently fatal, intracranial hemorrhages.

Central Birth Paralysis
Cerebral Hemorrhage   Apoplexia Neonatorum

Usually the seat of the hemorrhage is the subarachnoid space; often the delicate pia mater; sometimes between the dura and arachnoid;

more rarely between the meninges of the cerebellum; the lateral ventricles, and exceptionally the brain substance.

According to Seitz the hemorrhage is the result of rupture of the longitudinal sinus or veins, of the transverse sinus or of vessels of the chorioid plexus.

Fig. 49.—Method of insertion of trocar through the anterior fontanel to reach the ventricles. (After P. Ravaut.)

The symptoms differ with the extent and seat of the hemorrhage. For several hours no characteristic symptoms may be evident. However, most infants are born asphyxiated. The majority of those born alive succumb within a few days under symptoms of asphyxia and ate-

lectasis, slow, full, irregular pulse, frequently high fever, nystagmus, bulging of the fontanelles, sopor, convulsions, rigidity and paralysis. Those few who survive, often at an early age or later present the symptom-complex of cerebral paralysis (see p. 601) with or without idiocy.

The treatment is essentially the same as in traumatic cerebral hemorrhage in older children—principally surgical. (See p. 604.) An attempt may be made to relieve the intracranial pressure by lumbar puncture or aspiration of the subdural space. Lumbar puncture is always worth trying, and if it brings blood, which is often the case in infratentorial hemorrhage, the puncture should be repeated two or three times,* each time withdrawing from 5 c.c. to 15 c.c. Aspiration of the subdural space is accomplished by introducing a trocar almost parallel with the surface of the skull, at a point corresponding with the lateral angle of the anterior fontanelle, and withdrawing a sufficient amount of blood to relieve the pressure symptoms (Fig. 49).

F. C. Rodda† suggests the subcutaneous injection of 25 c.c. of blood, if the patient's blood is found wanting in coagulability.

Peripheral Birth Paralysis
Facial Palsy

Facial paralysis in the newborn is usually of traumatic origin as a result of pressure exerted upon the facial nerve by the obstetrical
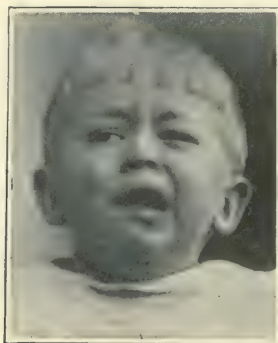
Fig. 50.—Obstetric facial paralysis in boy fifteen months old, which failed to yield to treatment.

forceps or deformed pelvis. It may be unilateral or bilateral. It resembles facial paralysis of older children (see p. 663) except that it

*H. Vignes, Progres Méd., No. 33, 1918, J. M. Brady, Jour. Am. Med. Assn., Sept. 21, 1918.
†Jour. Am. Med. Assn., Aug. 14, 1920.

runs a milder course. Very rarely the paralysis is permanent. It is important to differentiate this form of facial paralysis from that of central origin. In the latter form, as a rule, other portions of the body are involved, while the orbicularis palpebrarum remains free. The so-called congenital, nontraumatic facial paralysis is probably syphilitic in nature.

Brachial Paralysis—Obstetrical Paralysis—Duchenne-Erb Paralysis

In mild form it is of quite frequent occurrence. In typical cases the paralysis is usually limited (80 per cent) to the muscles supplied

Fig. 51.—Bilateral obstetric brachial paralysis, the so-called ''Duchenne-Erb Paralysis''.

by the brachial plexus composed of the lower four cervical nerves and the first dorsal, and their branches, *i. e.*, the deltoid, biceps, brachialis anticus, infraspinatus, supinator longus and the supinator brevis.

The arm (rarely both sides are affected—from reckless instrumental manipulations) hangs motionless, the upper arm is rotated inward,

the forearm is pronated, and the palm of the hand is turned backward and outward (Fig. 51). The wrist and finger-joints are usually only slightly affected; sensibility is intact and electrical reaction diminished or lost.

Recovery is the rule in mild cases. Those lasting over three months show trophic changes in the affected muscles, especially in the deltoid. The prognosis in cases of brachial paralysis presenting reaction of degeneration, is doubtful.

Fig. 52.—Obstetric brachial palsy: Erb's "upper arm type"; failed to respond to treatment.

Treatment.—After keeping the affected arm perfectly at rest for two weeks, the faradic or galvanic current should then be applied daily, for about five minutes at a time, until muscular power has been restored. Gentle massage and passive motion are very useful as a prophylactic against atrophy and contractures. In complete rupture of one or more cords of the brachial plexus, nerve end-to-end anastomosis and tendon transplantation are the only curative means at our command. Muscle training is indicated in children old enough to respond to suggestions.

II. DISEASES OF THE NEWBORN

Feeble Vitality of the Newborn

The physician is often confronted by a group of clinical phenomena in the newborn which may briefly be designated as "feeble vitality." It is a clinical entity which, though greatly at variance as to cause and ultimate course, presents at birth a uniform symptom-complex and demands a more or less uniform mode of treatment.

It is characterized by pronounced respiratory and circulatory disturbances, subnormal temperature, somnolence, general debility with or without emaciation, and is usually associated with one or several presently to be described diseased conditions.

1. Asphyxia Neonatorum

(SUSPENDED ANIMATION)

The asphyxia may be momentary, or last several minutes up to an hour or longer. Mild forms of asphyxia are manifested by slight lividity (asphyxia livida) of the face, feeble superficial breathing, and slow and weak heart beat. If the asphyxia is allowed to continue, the face becomes deeply cyanosed and congested, the eyes bulge, the muscular tonus and cutaneous sensibility are retarded, the umbilical cord is collapsed, and respiration is barely perceptible. Finally, the infant becomes deathly pale (asphyxia pallida), the muscular tonus and reflexes are lost, the heart beat is scarcely audible and respiration ceases.

Postmortem examination reveals overdistention of the right ventricle of the heart; cerebral, pulmonary and hepatic congestion; increased fluidity of the blood; serosanguinolent exudation in the serous cavities; accumulation of liquor amnii, blood and mucus in the air passages, and pulmonary atelectasis.

Prompt and prolonged resuscitating efforts (Sylvester's, Schultze's and Laborde's) are usually attended by favorable results. However, intracranial hemorrhage with consecutive mental and physical defects are not infrequent sequelæ of severe forms of asphyxia.

2. Atelectasis Neonatorum

(CONGENITAL COLLAPSE OF THE LUNGS)

Inflation of the lungs of the normal newborn begins with its first cry uttered announcing its arrival into the domain of the living. Succeeding respiratory acts gradually unfold the originally collapsed alveoli and bronchioles, and full expansion of the lungs is ordinarily

completed within the first forty-eight hours. The posterior portions of the lower lobes, particularly the right, are last to expand.

Failure of the lungs fully to unfold gives rise to the condition under discussion, *i. e.*, atelectasis pulmonum.

Most alveoli and bronchioles are collapsed. The lung is brownish red in color, feels tough and resistant to the touch—like liver—does not crepitate, and sinks in water. Usually both lungs, particularly the posterior parts of the lower lobes, are affected. In cases succumbing to the disease after weeks or months there is also found congestion of the heart, spleen and liver.

The causes of atelectasis are essentially the same as those of asphyxia; the former is sometimes a sequel of the latter, especially if inadequately treated. Inflation of the lungs is occasionally interfered with by congenital hyperplasia of the thyroid or thymus glands compressing the trachea.

In marked atelectasis the infant makes but faint efforts to respire. It is pale, sometimes cyanotic; its temperature is subnormal and its pulse slow and weak. It is unable to suckle properly and to cry aloud. It sleeps most of the time and but lazily responds to external influence. Auscultation discloses weak and vesicular breathing (never bronchial) and occasional crepitation. Slight dullness on percussion. The diagnosis may often be verified by a radiogram.

A great number of otherwise healthy children recover under prompt and energetic treatment. Delicate infants either die a few hours, days or several weeks after birth from prostration following repeated attacks of cyanosis, or survive and remain debile for life, often suffering from organic defects, such as incomplete closure of the foramen ovale or ductus arteriosus, and the like.

The treatment of atelectasis consists in stimulating the respiratory and circulatory functions by keeping the infant wide awake at intervals; frequent change of position; artificial respiration; alternating warm and cold baths or showers followed by brisk friction; oxygen inhalation, and gentle faradization. In all other respects they should be treated like premature babies.

3. Vitia Cordis

4. Syphilis Embryonalis S. Fetalis

5. Premature Birth

Children born before full term—between the twenty-eighth and thirty-eighth weeks of intrauterine life—are designated "premature."

Thanks to the earlier and better recognition of syphilis, the more thorough appreciation of the methods of its prevention and cure, as well as the tendency of the syphilitic virus spontaneously to lose its virulence through attenuation, premature births, being due chiefly to parental syphilis, are no longer as frequent in occurrence as in former years.

The physical condition of premature infants rests largely upon the period of prematurity, inherent vigor of the newborn, and the presence or absence of serious organic defects. Ordinarily premature infants are considerably punier than full term infants. They weigh and measure approximately—

			WEIGHT			SIZE		
At 29 weeks	....	1600 Gm.	3¼ lb.	40 Cm.	15	inches
" 31 "	....	1900 "	4 "	43 "	16¼	inches
" 33 "	....	2100 "	4¼ "	44 "	16½	inches
" 35 "	....	2600 "	5¼ "	47 "	17¾	inches
" 37 "	....	2800 "	5¾ "	48 "	18	inches
" 40 "	(full term).......	3100 "	6¼ "	52 "	19½	inches

The body is limp; the movements of the extremities are helpless and tardy. The face is usually sunken and senile. The skin is soft and delicate, vulnerable to an extreme, hence readily susceptible to infectious processes. Respiration is irregular, superficial and sometimes of the Cheyne-Stokes type. Atelectasis and cyanosis are not rare accompaniments. The heart beat and pulse are weak, often irregular, and the blood lacks in coagulating power. The bones are soft, more or less yielding to light manipulation. The temperature is subnormal. Premature infants, as a rule, are unable to suckle or swallow properly and, owing to incapacity of the digestive organs and atony of the intestinal musculature, fully to assimilate the food consumed. Severe colic and uric acid infarcts, which latter often lead to anuria and other uremic manifestations, add misery to their painful existence.

Encumbered with so many deficiencies, the span of life of the delicate premature infant must obviously measure but a few hours or days. The mortality of premature infants under 1,600 grams in weight, especially if they are inadequately cared for, is estimated to be about 80 per cent; of those weighing over 2,000 grams, 40 per cent; while of those weighing over 2,500 grams only 20 per cent—almost as low as with full-term babies. Such as survive, however, often remain very feeble for many years, manifest a greater tendency to disease, and lack power of resistance to overcome it. Occasionally, after many ups and downs, premature infants marvelously extricate themselves from the pangs of death and grow up full of vivacity and vigor. I have now

under observation a premature baby ten months old, weighing 12 lbs. that at birth weighed only $2\frac{1}{4}$ lbs. For the first six weeks it was fed on breast milk by means of a catheter through the nose.

It is therefore incumbent upon the physician to look upon every premature infant that respires at birth as one whose life can be preserved by suitable care and treatment.

Management of "Feeble Vitality of the Newborn" With Special Reference to the Premature Baby

Three special indications are to be met in the management of the newborn, who are delicate. We must (1) endeavor to maintain the

Fig. 53.—Incubator room for newly born babies with feeble vitality. (After Th. Escherich.)

best features of antenatal life; (2) supply nutriment suitable for the infant's growth and development; and (3) awaken and strengthen the dormant or inefficient functions of its organs.

The first prerequisite should be met by an artificial environment which should as nearly as possible resemble that of the interior of the uterus. In very delicate and puny babies the numerous modern incubators on the market, in many instances, answer the purpose.

The temperature of the incubator is maintained steadily at about 96° F., and fresh air supplied by the automatic ventilating contrivance and by, off and on, leaving the door open. Infants showing a fair amount of vitality usually get along very well without incubators,

the latter being supplanted by ordinary bassenets and warm-water bags, or preferably the modern electric pads. The infant is clothed in a woolen shirt and napkin and placed in the incubator or is wrapped in a ''premature gown'' which consists of a layer of absorbent cotton between two layers of gauze. A hood of the same material is attached to the body of the gown. The temperature of the baby's room should range between 74 to 78° F., or higher if the baby's temperature continues subnormal.

Delicate incubator babies should be disturbed as little as possible, and removed only for feeding and cleansing (by means of lukewarm oil) or for such therapeutic purposes (*e. g.*, artificial respiration, as necessity arises). Bathing is contraindicated, and any undue handling of the skin or mucous membranes must be carefully avoided, since most trifling injuries are very apt to be followed by fatal sepsis.

Every effort should be made to feed the premature infant on woman's milk for at least the first few weeks of extrauterine life. When too weak to suckle from the breast, the milk may be given every three
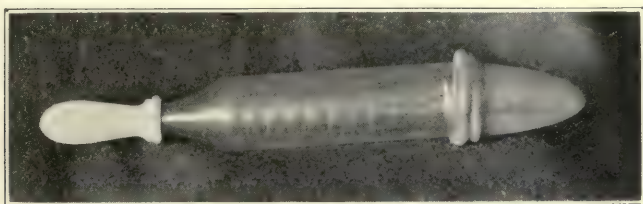
Fig. 54.—Breck's feeder

hours by means of a dropper or Breck's feeder, care being taken that the milk flows down into the throat very slowly, lest it enter the trachea and lead to aspiration pneumonia. In the absence of breast milk, light mixtures of cow's milk (¾ per cent of fat, ¾ per cent protein, and 6 per cent of milk sugar) should be administered every two to three hours in quantities of 4 to 8 teaspoonfuls. The amount is gradually to be increased. If the baby is unable to swallow, the milk may be administered by gavage, or by catheter through the nose.

The third indication applies principally to infants who, though born at full term, possess very little vitality, and whose organs, especially the heart and lungs, fail to functionate. The vitality is best aroused by artificial respiration—by alternate flexion and extension of the infant's body while it lies upon the operator's palms. An occasional dash of cold water upon its face, to induce the child to cry aloud and to take deep breaths, and stimulation by means of oxygen, caffeine and digitalis serve as useful adjuvants.

Sclerema Neonatorum

(Sclerema Adiposum)

This very rare affection may be primary, without any apparent cause, or secondary in nature as a result of great loss of body fluids (internal hemorrhages, gastrointestinal disease) or extensive exudations into internal cavities (thorax). It occurs principally in the premature, very feeble and badly nourished infants in the first few days of life, but also very much later, up to six months of age.

It begins in the lower extremities, particularly the calves. From here it spreads symmetrically over the thighs, loins, trunk, neck, upper extremities and head, leaving penis, scrotum, planta pedis, and palma manus uninvolved. The skin is dirty yellow, very tense, cold, hard, immovable over the underlying structures, and does not pit on pressure.

From day to day the skin becomes more indurated, marbleized, and the patient lies stiff with rigid, mask-like face and firmly closed mouth as though in a state of tetanus. Sucking is often impossible. There is gradual sinking of all vital functions. The temperature falls (to 85° F., or lower), the heart action becomes weak, the pulse is slow and barely perceptible, respiration shallow and irregular, the voice feeble and whining, the intestines and kidneys are inactive, the child wastes rapidly and death ensues in about a week from exhaustion or from some complication, the commonest being pneumonia and sepsis. Milder cases, especially older infants, not infrequently recover.

Treatment.—Early hypodermo- and entero-clysis with hot (104° to 106° F.), normal saline solution (from 2 to 3 ounces t. i. d.); gentle massage with oil; stimulation; maintenance of body heat; careful feeding, etc., as outlined under ''Feeble Vitality of the Newborn.'' (See p. 216.)

Scleredema Neonatorum

(Sclerema Serosum)

This form of edema affects especially premature, weak (twins), atelectatic and syphilitic infants. It usually begins a few days postpartum (it is rarely congenital) with puffiness and swelling of the feet and legs. The edema soon extends upward (involving also the mons veneris, scrotum and penis) over the entire body except the chest, and rarely the eyelids and face. The skin is tense, shiny, waxy white, or cyanotic, and pits on pressure. When the edema increases it greatly resembles true sclerema, but may be differentiated from the latter by bearing in mind the following characteristic symptoms:

	SCLEREMA	SCLEREDEMA
Color of skin	Dirty yellow.	Shiny or mottled.
Parts exempt..................	Genitals, palms of the hands and soles of the feet.	Chest.
Pitting on pressure.............	Absent.	Marked.

The general symptoms, such as low temperature, great depression, etc., are not quite as pronounced as in sclerema adiposum.

The prognosis is not as grave as in true sclerema.

The treatment consists chiefly of stimulation (camphor, digitalis) hot baths, massage and passive motion, active diuresis and proper feeding. (See also ''Feeble Vitality of the Newborn,'' p. 216.)

Sepsis Neonatorum

With the usual aseptic precautions that are now being taken in the management of labor and the puerperium, the number of cases of sepsis neonatorum has been reduced to a minimum. This is true especially of systemic sepsis. The extreme importance, however, of the subject in question, demands its careful consideration.

LOCAL SEPSIS

Omphalitis (Inflammation of the Navel)

Simple omphalitis is manifested by delayed closure of the umbilical wound after separation of the umbilical cord, wetness, slight suppuration, and incrustation. There is no inflammatory reaction in the surrounding parts. The general health is undisturbed.

Phlegmonous omphalitis usually begins the second week after birth. The navel forms an ulcerated conical projection. The surrounding tissue is firm, infiltrated, glossy and painful to the touch. Sometimes the inflammation extends rapidly over the abdominal wall or into the deeper structures, giving rise to peritonitis. In one case under observation, secondary suppurative foci developed in the lower portion of the gladiolus sterni (leaving behind an open fistula) and in the left hip-joint, completely destroying the caput femoris and giving rise to a permanent dislocation (Fig. 55). The constitutional symptoms vary with the degree of the severity of the affection, but are sufficiently pronounced to make the child quite ill and to render the prognosis doubtful. Milder cases may terminate in suppuration, but with careful treatment (see p. 221) end in recovery.

Fig. 55.—Absorption of left head of femur and consequent dislocation of the hip in a child two years old as a direct result of sepsis neonatorum which began with an infection in the navel.

Erysipelatoid omphalitis is a very grave affection, often terminating fatally either within a few days from exhaustion or a week to ten days later from septic peritonitis, icterus, and local suppuration. The symptoms and treatment are the same as in ordinary erysipelas.

Diphtheritic omphalitis (ulcus umbilici) is characterized by a fibrinous umbilical exudation which, when cast off, leaves behind a superficial or deep ulcer. Occasionally it is due to the Klebs-Löffler bacillus.

Gangrenous omphalitis ends fatally in the majority of cases. At first a small, discolored, ulcerated spot, if not immediately arrested, it rapidly develops into a large, gangrenous, fetid mass. It sometimes extends into the deeper structures, giving rise to peritonitis, urinary and fecal fistulæ, profuse hemorrhage and pronounced constitutional symptoms.

Treatment.—As the umbilical wound forms the principal and most frequent portal of entry for septic infection, the importance of caring for the umbilicus with the minutest detail is quite obvious. Strictest cleanliness should be enforced and unnecessary handling prohibited. Clean scissors, clean ligature, preferably composed of several strands of cotton or silk thread, and, above all, clean hands should be used in cutting, ligating and dressing the cord. The dressing should consist of a few layers of sterile linen cloths and dusting powder (1 part of salicylic acid and 6 parts of starch) and be changed every alternate day, preceded by cleansing the wound with a little pure alcohol to hasten desiccation of the umbilical rest. As moisture favors the growth and absorption of the bacteria which accumulate at the naval wound, the child should receive daily a sponge bath instead of a tub bath, until the navel has completely cicatrized.

To prevent hernia as well as access of dirt, the umbilical band should be continued for a few weeks after complete healing of the navel.

If inflammation of the navel, no matter how slight in degree, occurs notwithstanding all the precautions, it should receive immediate and energetic treatment. Procrastination is dangerous, nay, often fatal.

Cauterization of the affected parts with a 2 per cent to 5 per cent solution of nitrate of silver, once a day or less often, is very useful in all forms of omphalitis. The wound should be kept scrupulously clean, and protected by a moist (boric acid, 4 per cent solution) gauze dressing, covered by rubber tissue. If the septic process does not yield to this treatment early, a surgeon should be consulted. A bacteriologic examination may prove helpful in giving a correct clue as to the treatment, as for example, in diphtheritic omphalitis, where diphtheria antitoxin is of undoubted benefit. (See "Biologic Therapeutics," pp. 75 and 82.)

Omphalorrhagia (Bleeding from the Navel—Idiopathic Umbilical Hemorrhage)

Umbilical hemorrhage may occur as a result of tearing the cord during delivery, defective ligation, or imperfect establishment of respiration (delaying the closure of the umbilical vessels). The hemorrhage may be slight or severe, but is readily controllable. In contradistinction to these forms of navel bleeding which take place soon after birth, there is another variety of bleeding from the navel, the so-called *idiopathic or spontaneous umbilical hemorrhage* which occurs at about the time the umbilical rest separates (between the fourth and ninth days). The bleeding takes the form of a steady oozing of blood as though coming from a compressed wet sponge. It is probably due to sepsis of the umbilical blood vessels. Some authors are inclined to attribute it to congenital syphilis or transitory hemophilia. (See p. 229.) In a great many instances the hemorrhage cannot be arrested, death taking place either from exsanguination or from gradual exhaustion and complications (sepsis).

For details of treatment see "Melena."

Umbilical Granuloma (Excrescence, Fungus, Sarcomphalos)

It is a strawberry-like, small tumor, attached to a broad base or pedicle at the umbilical stump. It bleeds readily and usually discharges thin pus. Like exuberant granulations in other localities, it is promptly cured by a few applications of nitrate of silver (the stick, or 10 per cent solution). It should not be confounded with "Persistent Omphalomesentericus."

Ophthalmoblennorrhea Neonatorum (Gonorrheal or Purulent Ophthalmia)

Gonorrheal ophthalmia is caused by infection of the conjunctiva of one or both eyes by the Neisser gonococcus. The inoculation usually occurs during the passage of the head through the parturient canal containing a gonorrheal discharge. It may also be conveyed to the eyes of the infant postpartum by means of the fingers of the attendant or articles in use which have been soiled by the purulent discharge.

The disease begins two or three days after the gonorrheal inoculation, with intense tumefaction of the lids, redness, swelling and thickening of the conjunctivæ, lacrimation, and mucous and mucopurulent secretion. From day to day the discharge becomes thicker and more purulent; the conjunctiva assumes a velvet-like appearance (chemo-

sis), and papillary deposits or longitudinal folds appear upon the conjunctiva bulbi. If not immediately arrested, especially if the purulent secretion is allowed to accumulate between the edematous, pasted lids, the disease spreads rapidly to the cornea causing haziness, maceration and partial or total perforation. As a result of the latter and depending upon its location, total or partial staphyloma, panophthalmitis, with phthisis bulbi, capsular cataract, and anterior synechiæ may supervene.

Occasionally, particularly in delicate infants, gonorrheal conjunctivitis gives rise to numerous complications, such as articular affections, gonorrheal rhinitis, stomatitis, etc.

The duration of the disease varies from four to eight weeks.

Until the introduction of Credé's method of prophylaxis, gonorrheal ophthalmia was supposed to have contributed 60 per cent of the cases of blindness of one or both eyes. At present the percentage has been reduced to one-third, and with early and careful treatment the prognosis is still more favorable.

PROPORTION OF PUPILS NEWLY ADMITTED TO NEW YORK SCHOOLS FOR THE BLIND DURING THE PAST TEN YEARS WHO ARE BLIND FROM OPHTHALMIA NEONATORUM

School Year	No. of public schools	Total new admissions	Blind from O. N.	Per cent
1907-08.........	10	290	77	26.5
1908-09.........	14	300	68	22.6
1909-10.........	13	325	67	20.6
1910-11.........	15	351	84	23.9
1911-12.........	24	415	88	21.2
1912-13.........	21	386	88	22.7
1913-14.........	19	428	84	19.6
1914-15........	28	602	91	15.1
1915-16........	35	666	127	19.0
1916-17........	34	647	119	18.4

Gonorrheal ophthalmia is not to be confounded with simple conjunctivitis not infrequently met in the newborn in connection with local sepsis. The latter variety is readily recognized by the absence of gonococci in the discharge and by its much milder course.

Treatment.—Where there is the least suspicion of gonorrhea in the mother, her parturient canal and external genitalia should be carefully disinfected by a bichlorid solution (1 to 5000) before, during, and after delivery. In addition to this, the following directions in the way of prophylaxis (Credé's method) should be promptly resorted to: Wash off each eye with a boric acid wipe; into each eye instill two drops of a 2

per cent solution of silver nitrate; in about thirty seconds wash out the excess with saline solution. This should be done as early after birth as possible. During the puerperal state the child should be kept away from the mother.

If only one eye be affected the fellow eye should be securely covered by a watch-glass or a small pad of lint, oiled silk and roller bandage. This protected eye should be inspected and cleansed twice daily.

As soon as the child is seen by the physician, he should pencil the affected eye with a 2 per cent silver solution. If this occurs early, the ophthalmia may sometimes be arrested in its incipiency or at least rendered milder in its course.

The affected eye must be handled by the nurse from behind the patient's head. Small, round layers of lint are transferred, every three to five minutes from a large square of ice to the affected eye, continuously for one hour. An intermission of one hour is then given and the cold applications are resumed. This should be continued day and night until there is positive evidence of abatement of the inflammation and excretion. This usually occurs within two weeks. The eyes should be carefully but very gently cleansed every half hour with warm, saturated solution of boric acid (4 per cent). If the lids are so swollen as not to permit thorough cleansing, canthotomy may have to be resorted to. Silver being the most proficient antigonococcus, a 2 to 3 per cent solution should be applied to the conjunctiva daily so long as the excretion is profuse and less often when it becomes more scanty and less purulent. Instead of nitrate of silver we may employ argyrol, silvol, solargentum, or protargol in from 5 per cent to 10 per cent solutions. In involvement of the cornea the ice cloths should be discontinued, and warm applications used instead. A 1 per cent solution of atropine should be used as necessity arises. In bad cases antigonococcus vaccine is worth trying.

Examination of the discharge for gonococci should be made at least once a week, and the case should not be regarded noncontagious and out of danger until the discharge from the eye remains free from gonococci for at least two weeks. The treatment of gonorrheal ophthalmia should not be intrusted to unskillful hands. The better trained the nurse is in handling serious eye cases, the more rapid and perfect the recovery.

Pemphigus Neonatorum

Simple, nonsyphilitic pemphigus (see p. 484) makes its appearance between the fifth and twentieth day of the child's life. It is quite communicable, sometimes epidemic, and is probably due to the staphy-

lococcus pyogenes aureus. Its seat of predilection is the abdomen and inguinal region, but the lesion may be found on any part of the body. It but very rarely affects the palms of the hands and the soles of the feet, herein markedly differing from syphilitic pemphigus. The eruption consists of tense bullæ, varying in size from a lentil to a quarter of a dollar piece and contains a serous, rarely seropurulent fluid. The blebs are situated upon a reddened base, and on bursting leave moist, red spots which very soon are covered over by skin. Occasionally ulceration of the skin supervenes, and is accompanied by high fever and other constitutional symptoms (malignant pemphigus). This severe form of the disease is observed particularly in cachectic and bottle-fed infants exposed to insanitary surroundings, and often leads to a fatal issue. In otherwise healthy, well-nourished and well-kept infants, recovery may be expected within from two to three weeks.

Simple pemphigus is preventable by strict attention to general hygiene and proper feeding. Those in charge of the child should be cautioned as to the communicability of the disease. If large surfaces are involved, warm baths are very useful, preferably with boric acid (2 per cent), solutions. They may be administered two or three times a day and followed by dusting over the moist surface with—

R̥ Bismuthi subgall.,
    Acidi salicyl ....āā gr. x. | 0.6
    Zinci stearat. .... ʒi | 30.0

and enveloping the body in cotton. Occasionally, applications of a 2 per cent solution of nitrate of silver. Autogenous vaccine in malignant cases.

Dermatitis Exfoliativa Neonatorum

Slight dermatitis, or erythema, with or without desquamation, is more or less physiologic in the newborn. There is, however, an obscure (sepsis?) form of exfoliative dermatitis which is peculiar to early infancy (usually in the second, rarely after the fifth week of life), and is closely related to pemphigus. It begins with inflammation of the oral mucous membrane, rhagades at the angles of the mouth, and diffuse redness of the entire body, followed by active desquamation of the skin in large lamellæ. It is sometimes preceded by detachment of skin and bursting of vesicles filled with clear fluid. Not infrequently the erosions extend to the oral mucous membrane.

The disease runs its (afebrile) course in a few weeks, and in robust children ends favorably. In delicate children it may be followed by

general furunculosis or even gangrene, gastrointestinal disturbances and pneumonia, and prove fatal.

Like nonsyphilitic pemphigus, dermatitis exfoliativa is preventable by scrupulous cleanliness, and the avoidance of local irritation. The local treatment consists of inunctions of 1 per cent salicylic or carbolic acid oil.

GENERAL SEPSIS

In speaking of primarily local septic affections, attention has been directed to the frequency with which grave constitutional symptoms are observed during their protracted course. In these cases the systemic manifestations are secondary to the local ones, and if the latter are detected and treated early, the former may be prevented or arrested in their incipiency. We are now about to describe a group of diseases in the newborn, which either present no visible local lesions at all, or are so slight as to escape attention in their early stages.

Tetanus (Trismus) Neonatorum

Tetanus in the newborn, like the corresponding disease in the adult, is due to the tetanus bacillus (Nicolaier, Kitasato). Infection usually occurs through the umbilical stump or circumcision wound. The bacillus multiplies by spore formation and generates toxins which enter the system and are absorbed principally by the ending of the motor nerves. From here the toxins are ultimately carried to the anterior horns of the spinal cord and the nuclei of the medulla oblongata—hence the tetanic contractions.

The symptoms begin within the first week after birth, or later after ritual circumcision, with restlessness, dropping of the nipple of the breast or bottle with a cry, and tension of the masseters. The spasm rapidly involves the orbicularis oris and palpebrarum muscles, the lower jaw becomes rigid, the mouth proboscidiform, the forehead and cheeks are wrinkled, and the eyelids are half closed (risus sardonicus). The hands are clenched, the legs flexed and abducted and, varying with the degree of severity of the attack, there is more or less marked opisthotonos. At first the paroxysms occur only during the act of nursing, gradually, however, more frequently and more persistently. In severe cases there are also spasms of the glottis, of the esophagus, and diaphragm, and in consequence attacks of asphyxia which may end fatally. On the other hand, the affection may run a protracted course, sometimes for weeks, and occasionally end in recovery.

The more violent the attacks and the higher the temperature, the less favorable the prognosis. Seventy per cent of the cases succumb within a few days, either from spasm of the diaphragm or, more rarely, from exhaustion.

Treatment.—Careful protection against wound infection and prompt attention to existing traumatism. Considering the very grave prognosis under the ordinary methods of treatment and the occasional success obtained by means of hypodermic or subdural administration of tetanus antitoxin, the latter should be resorted to at the earliest possible time, either as a prophylactic immediately after the injury

Fig. 56.—High degree of ''tetanism'' greatly resembling tetanus neonatorum. Note Fig. 59, showing same case during partial relaxation of the spasm.

(500 units) or as a curative measure (2,000 units p. r. n.), in addition to the symptomatic treatment generally in vogue.

The Department of Health of the City of New York suggests the following procedures in a developed case:

A lumbar puncture having been performed in the usual way, 1000 to 2000 units of antitoxin, heated to body temperature, are allowed to run into the spinal canal by gravity. In order that the antitoxin may be distributed throughout the length of the cord, it should, if necessary,

be diluted with sterile saline to a volume of at least 5 c.c. In case of a "dry tap," which has been reported on good authority as occasionally occurring in this disease, this amount should not be exceeded, but when an abundance of spinal fluid is obtained, the intraspinal dose should be little less than that of the fluid withdrawn. An intravenous injection of 3000 units should be given at the same time in order to render the blood highly antitoxic at once. The intraspinal injection may be repeated in 24 hours and again in 48 hours, but a third dose is probably unnecessary. A subcutaneous injection of 2000 units may also be given on the fourth day to sustain the antitoxic strength of the blood. If for any reason the attending physician is not able to give an intraspinal injection, an intravenous dose of 3,000 to 5,000 units should be given. If this, too, is impossible, rather than delay, the same dose or a larger one should be given intramuscularly, several muscles being used, and arrangements immediately made to give an intraspinal and intravenous dose at the earliest possible moment.

The use of antitoxin does not do away with the necessity for thorough surgical treatment of the wound if it has not already healed. The patient should be protected as carefully as possible from noise, excessive light, drafts, jars, and other forms of irritation. Any irritation of the skin should be avoided. To combat individual symptoms we may resort to lukewarm baths, choral hydrate, the bromides per rectum, and heart stimulants, especially camphor. Feeding (mother's or diluted cow's milk) with a soft rubber tube through the nose.

Arteritis and Phlebitis Umbilicalis

This condition is usually observed secondarily to omphalitis ($q. v.$), but may occur as a primary disease. In the latter event no local alterations are discernible at the navel, and the grave affection frequently escapes notice until pronounced symptoms of general sepsis make their appearance. These consist of restlessness, fever, prostration and death within a few days, or gradual exhaustion from numerous complications. In umbilical phlebitis intense icterus—from extension of the inflammation to the liver—forms a characteristic symptom. In some cases of arteritis and phlebitis umbilicalis a fistulous tract is observed at the navel which on pressure discharges blood and pus containing pathogenic microorganisms.

For prophylactic and local treatment see "Omphalitis" (p. 221). The constitutional symptoms call for symptomatic treatment. Thus, careful feeding, preferably breast milk; active stimulation by means of enteroclysis, hypodermoclysis, sterile camphorated oil, etc. Antistreptococcic serum is deserving of trial.

Erysipelas Neonatorum

This affection begins suddenly, with high fever, convulsions, and often other symptoms of general sepsis. The glossy redness rapidly extends over large areas, often over the entire body. The disease proves fatal in a few days, and the cases that survive the acute attack usually succumb to cutaneous necrosis (particularly of the scrotum, extremities), copious diarrhea, septic peritonitis, pneumonia, and exhaustion.

The treatment is principally prophylactic. The inflamed areas should once a day be painted with pure ichthyol, or kept moist with gauze saturated with a 50 per cent solution of Epsom salts. In one desperate case under our care the rapid spread of the inflammation was arrested by painting the affected parts with pure carbolic acid followed by sponging with absolute alcohol. Antistreptococcus serum.

Melena Neonatorum

Melena *vera* should not be mistaken for melena *spuria*, in which condition the blood originates from erosions in the mouth or nasopharynx or from swallowing of blood from fissured nipples, etc.

Melena vera usually begins in the first few days of the child's life with bleeding from the bowels, and often with hematemesis. As a rule, the blood is mixed with stool, and is dark brown or black in color. In some cases the loss of blood is slight, recurs at long intervals and terminates spontaneously without serious consequences except tedious convalescence. In the majority of cases of genuine melena, however, the bloody discharge is profuse and leads to rapidly increasing anemia and collapse. Eliminating the group of cases which are due to a hemophilic dyscrasia, authorities are not agreed on the actual cause of true melena. In a number of cases postmortem examination disclosed erosions and ulcerations of the stomach and intestines which are attributed to thrombosis of the umbilical vein or the ductus Botalli. The consensus of opinion, however, favors the septic origin of the necrosis erosions. (For treatment see p. 230.)

Epidemic Hemoglobinuria With Icterus in the Newborn

(Cyanosis Icterica Cum Hemoglobinuria, Winckel's Disease)

This extremely grave (90 per cent mortality) epidemic affection makes its appearance about the fourth day postpartum, in apparently healthy-born and well-developed children. The infant becomes restless, refuses nourishment, shows signs of respiratory disturbance and slight rise of temperature. The skin turns greenish yellow, and soon

deeply jaundiced and cyanotic. Collapse, somnolence and convulsions, rarely preceded also by vomiting and diarrhea (no blood), are rapidly followed by death. The urine is pale brown, contains hemoglobin, renal epithelium, granular and blood casts, and masses of detritus, but no free blood corpuscles.

The autopsy reveals congestion and fatty degeneration of the internal organs, with punctiform hemorrhages, especially in the mucous and serous membranes; masses of granular hemoglobin in the kidneys and spleen and thickening of the blood.

Acute Fatty Degeneration of the Newborn (Buhl's Disease)

The essential anatomic features of this rare but very malignant affection are fatty degeneration of the internal organs, notably the heart, liver and kidneys, and hemorrhages in the viscera, and into the serous cavities.

The disease attacks full-term infants who for some inexplicable reason are born asphyxiated. Those few who survive, respire badly, are cyanotic, or rather icteric, and present hemorrhages in the skin and mucous membranes, from the alimentary canal, and the umbilicus. They almost invariably succumb before the end of the second week from progressive anemia, anasarca, and collapse.

Treatment.—The indications for the treatment of any of the afore mentioned hemorrhagic manifestations are: (1) to arrest the hemorrhage; (2) to improve or at least, maintain the vitality of the newborn infant. In former years considerable reliance was placed on a number of local hemostatics to arrest the hemorrhage, chiefly the actual cautery, adrenalin, perchloride of iron, ice and compression; for a time also calcium chloride internally and sterile, warm gelatine (10 per cent solution, 2 to 5 drams t. i. d.) hypodermically. Nowadays, however, all these doubtful procedures have been practically abandoned.

Whether the hemorrhage be due to congenital hemophilia or sepsis, the best results are obtainable from subcutaneous injection either of blood serum 10 to 20 c.c. or whole blood (10 c.c. to 30 c.c. to be withdrawn from the vein at the bend of the elbow of a donor or parent), or both, after a short interval; or from direct transfusion. This last method is especially indicated in hemorrhage associated with sepsis; but because of the extreme difficulty of doing a transfusion on a newborn, owing to the minuteness of the blood vessels, the operation should be performed by an expert. It has recently been shown that in infants the longitudinal sinus serves as an excellent, safe and easily accessible route for transfusion. The baby is immobilized as for intubation, the head is steadied by an assistant, and the sinus is

reached by introducing (1 or 2 millimeters deep) a needle, 20 or 22 gauge, one-half inch long. The injection of human serum or whole blood may be repeated every four to eight hours. Where human blood serum is not obtainable, horse or rabbit serum may be used instead. Transfusion may be performed by end-to-end anastomosis, by the Lewiston method of citrated blood, or by the direct Unger method.

To meet the second indication the reader is referred to the instructions given under the "Management of Feeble Vitality of the Newborn," p. 216.

FUNCTIONAL DISORDERS OF THE NEWBORN

(URIC ACID INFARCT, ICTERUS, MASTITIS)

Uric Acid Infarct

The urine of the newborn is clear immediately after birth, but turns turbid soon after and remains so for the first four or five days. It contains bladder and kidney epithelia, hyaline and epithelial casts, and a large quantity of urates. In consequence of the sudden alteration in the blood circulation there is an excessive excretion of nitrogenous metabolic products, and as the newborn consumes but very little water during the first few days of life, uric acid crystals and ammonium urate, instead of being washed away, are retained in the renal tubules.

The symptoms accruing from this functional insufficiency depend greatly upon the degree of obstruction of the urinary tubules. Ordinarily, gradual elimination of the uric acid and ammonium urate crystals occurs within a few days without any abnormal manifestations, except restlessness and crying just before and during the act of urination, and passage of small quantities of highly colored urine showing brick-red stains and a fine granular deposit on the diaper. Occasionally however, we find complete retention of urine, fever, and, owing to irritation of the renal pelvis, nephritis with its concomitant symptoms (albuminuria neonatorum).

Treatment.—Large quantities of fluids, hot baths, mild diuretics.

℞ Kalii acetatis .... ʒss.	2.0
Aq. fœniculi .... ℥iij.	100.0
M.	
S.—ʒi every hour if necessary.	

Icterus Neonatorum Catarrhalis

The theories promulgated to explain the causation of icterus in the newborn are so numerous, pedantic and contradictory, that for the

sake of clearness, they are best left alone. It is perfectly logical to
look upon this common (in about 80 per cent of all newborn infants)
and harmless phenomenon as an expression of the active physiologic
changes in the liver to which all other organs are subjected in the
first few days of life. Hess believes the condition to be due to a con-
gestion of the biliary capillaries resulting from an insufficient ex-
cretion of bile into the duodenum. It would seem, however, plausible
to assume that analogous to catarrhal jaundice in older children, icterus
of the newborn is also a manifestation of gastrointestinal irritation,
produced by the sudden demand upon the digestive system to exer-
cise functions hitherto not accustomed to.

The yellowish discoloration of the skin usually appears on the second
or third day on the face and chest and gradually extends to the ab-
domen and extremities and, rarely, also to the scleræ. The icterus
runs an afebrile, uncomplicated course of about two weeks' duration.
Cases of a more protracted course and presenting more or less severe
general symptoms should always be looked upon as a partial manifes-
tation of sepsis neonatorum. They may also be due to congenital
syphilis, congenital obliteration of the bile ducts (q. v.), or possibly
also to congenital cirrhosis of the liver.

Mastitis Neonatorum

Moderate swelling of the mammary glands of the newborn and dis-
charge of a milk-like secretion (''witch's milk'') is physiologic in in-
fants of both sexes. It begins between the first and third weeks of
life and may persist for weeks without giving rise to ill effects. Oc-
casionally, however, as a result of traumatism or infection, it may
terminate in acute inflammation or even suppuration. In this event
the breasts are red, swollen and painful, and may present fluctuation
at one or more points, and constitutional symptoms, such as restless-
ness, vomiting, and fever.

If the mammary glands are from the beginning not subjected to
meddlesome interference, in short, are left entirely alone, there is
usually spontaneous, gradual *restitutio ad integrum*. Should inflamma-
tion ensue, the breasts should be wrapped in oiled cloths or absorbent
cotton or gauze saturated with a 2 per cent boric acid or bichloride
solution (1:10000), lightly painted with tincture of iodine, or covered
with emplastrum belladonnæ smeared on soft thin leather. In the
event of suppuration, if not relieved by spontaneous evacuation of
the pus, a radiate incision under aseptic precautions is indispensable.

Phlegmonous inflammation and gangrene are rare complications.
while atrophy of the mammary glands and more or less loss of func-
tion may prove very serious to girls.

CHAPTER V

DISEASES OF THE ALIMENTARY TRACT

DISEASES OF THE MOUTH

Stomatitis

Stomatitis or inflammation of the mucous membrane of the oral cavity is a more or less contagious affection peculiar to infancy and early childhood. It varies in intensity from simple temporary catarrh to fatal gangrene. It is invariably of parasitic origin. The degree of severity of the disease depends upon the pathogenicity of the parasite, the power of resistance of the patient, and the promptness and accuracy of the treatment.

Stomatitis occurs principally at a time when the child's health is undermined, as, for example, during dentition, or synchronously with acute infectious diseases. Even normally the mouth forms a favorable nidus for cocci, bacilli, spirilla, leptothrix, and similar vegetations, and their growth is surely enhanced by allowing the child to enjoy its acrid nasal discharge; to suck on dirty nipples, toys, and eatables; by keeping its mouth and teeth filthy; by denuding the oral mucous membrane of its epithelium by brisk rubbing in the act of cleansing, and by permitting every friend or kin to infect the child's mouth by overindulgence in the art of osculation. Finally, dental caries, hemorrhagic affections, intoxication from the use of mercury, bismuth, etc., among many other diseased conditions, frequently form contributing causes of stomatitis.

In accordance with the seat and appearance of the lesion it is customary to distinguish the following varieties of the disease

1. **Stomatitis Catarrhalis (Erythematosa).**—Redness and slight tumefaction of several portions of the mucous membrane of the mouth, coated tongue with prominent papillæ and red tip and edges. Often marked salivation.

2. **Stomatitis Mycotica (Soor, Thrush, Sprue).**—Probably due to a hyphomycete, the *Monilia candida*. Usually begins with a fine, white, flour- or casein-like deposit upon the slightly reddened tongue and buccal mucous membrane. The deposit may be yellowish or blackish in color. If not arrested, the dots and maculæ coalesce and often extend to the pharynx, esophagus, stomach and intestines. This is apt to occur especially in atrophic children.

3. **Stomatitis Maculofibrinosa (Aphthosa, Follicularis, Herpetiformis).** —The causal microorganism is still undetermined. Often begins with small vesicles. The inflamed mucous membrane is here and there (usually the anterior part of the mouth) covered with small, grain- to lentil-sized, variously shaped, yellow, grayish-yellow, or grayish-white foci surrounded by a dark-red areola. By coalescence of several follicles, large raised plaques are sometimes observed. *Fetor ex ore.*

4. **Stomatitis Ulcerosa (Stomacace).**—It is attributed to the *Bacillus fusiformis* and the *Spirochaete denticola.* The lesion consists of numerous, grayish, irregular, ulcers with a bleeding base and angry-looking areola, situated at first on the red, spongy and painful gums, and, if not arrested, spreading to the tongue, cheeks or lips and tonsils. *Fetor ex ore.* In bad cases also the teeth loosen and the lymph nodes swell.

This form of stomatitis differs, from the yellowish to greenish, superficial, easily bleeding ulcers, known as *Bednar's aphthæ* (ulcera pterygoidea), of the newborn or young infant, by the fact that the latter appear symmetrically on each side of the median raphé near the junction of the hard and soft palates, and are usually the result of abrasion of the epithelium by too strenuous cleansing of the mouth.

It may occasionally also be mistaken for the exceptionally ulcerating, so-called *"epithelial pearls."* These innocent milia-like dots, however, are usually found only in the newborn, and situated along both sides of the raphé of the palate.

5. **Stomatitis Gangrenosa (Noma Faciei, Cancrum Oris).**—It occurs principally in cachectic children, chiefly between two and five years old. It may follow ulcerative stomatitis or acute exanthematous diseases (measles!) and begins with a small, rapidly spreading brownish, greenish ulcer upon a hard, elevated base, on the inner surface of the cheek, near the angle of the mouth or on the lips. Very soon a black spot appears on the outside of the cheek, surrounded by marked tumefaction of that side of the face and the submaxillary glands. The cheek becomes perforated, the edges of the wound turn black, and the sloughing process spreads rapidly so that the whole thickness of the cheek has the appearance of a dirty, greasy scab, and within a few days may be completely destroyed. Also necrosis of the jaw and general toxemia. Rapid exhaustion.

In addition to these definite varieties of stomatitis, we occasionally meet with involvement of the oral mucous membrane as a result of

PLATE III

STOMATITIS APHTHOSA (ADVANCED STAGE)

(*Courtesy of Dr. John Zahorsky.*)

diphtheritic or gonorrheal infection, as also a pseudomembranous form arising from traumatism and subsequent streptococcic infection of the mucous membrane. This last variety is not rarely observed in the newborn, occasionally forming a partial manifestation of sepsis neonatorum (*q. v.*)

Mild or even moderately severe cases of stomatitis rarely give rise to systemic disturbance, and unless the local lesion is situated on the lips, tongue, or gums and interferes with sucking, or chewing, several days may pass before the disease is detected. Sometimes the patient is feverish and restless, cries and refuses food in the earliest stage of stomatitis, but the constitutional symptoms do not stand in direct ratio to the extent and gravity of the local manifestations. However, with persistence of the local symptoms, sooner or later the general health participates in the pathologic process. Starch digestion is greatly impaired by the excessive loss of saliva, which almost incessantly dribbles from the swollen, reddened, half-closed lips, and vomiting and severe diarrhea are frequent results of swallowing of the putrid saliva and the decomposing, more or less ichorous and membranous oral contents. These latter symptoms, in addition to the emaciation from refusal of food and absorption of septic material, greatly delay convalescence and may lead to gradual or rapid exhaustion and fatal issue. In the absence of such grave symptoms and with early and careful treatment, however, the prognosis is good in all forms of stomatitis, except noma (75 per cent mortality).

Treatment.—Above all, cleanliness should be enforced, and the sooner it is begun the surer we are of rendering the disease free from untoward consequences. Strictest cleanliness of the food, feeding-bottles and nipples, cups, spoons and everything else coming in contact with the child's mouth, should be observed. The child's mouth should be regularly washed after each feeding, by *gently wiping* it with absorbent cotton dipped in a 2 per cent watery solution of boric acid or bicarbonate of soda. As to general cleanliness, see "Hygiene," p. 64.

In mild cases it is usually sufficient to paint the affected parts once a day with a 2 per cent solution of nitrate of silver and to employ the following mouth wash every two to four hours:

℞ Acid borici,
      Sod. boratis ....āā ʒj. | 4.00
      Hydrogen dioxidi,
      Glycerini ....āā ℥j. | 30.00
      Alcoholis .... ʒiv. | 15.00
      Aq. rosae ....q. s. ℥iv. | 120.00
  M.
  S.—To be diluted with an equal quantity of water, as a mouth-wash.

Should the stomatitis fail to yield to the treatment after twenty-four to forty-eight hours, more energetic measures should then be adopted to stay its destructive tendencies. The strength of the silver solution should be doubled, and the mouth irrigated every two hours with 1 per cent permanganate of potash, 5 per cent Labarraque's solution, $\frac{1}{4}$ to 1 per cent of chlorazene or Chloramine T (Dakin's antiseptic), etc.

It is often advantageous to suspend milk feeding for a few days and to nourish the child on broths, light cocoa, cereals, toast and tea, pineapple juice, etc. Protracted illness demands active stimulation by means of good wines (diluted), strychnine, and compound tincture of cinchona. This may be combined with the rhubarb and soda mixture to remedy gastrointestinal disturbance which is ever present in cases of long standing. In the majority of instances even severe cases of stomatitis promptly respond to this mode of treatment. An exception to this rule is made, however, by *noma*,—that rapidly advancing form of necrosis, which knows no barrier to its destructive, death-dealing trail, and often even the knife fails to stay its ravages. At the earliest possible moment the gangrenous portion should be destroyed with the caustic stick, nitric acid or, preferably, with the actual cautery. Frequent cleansing of the parts should be continued day and night, and strengthening food and stimulants administered at short intervals. Since Loeffler's bacilli are found in a number of cases of noma faciei and vulvæ, diphtheria antitoxin (5 to 10,000 units) should be resorted to early in the course of the disease. Very often everything fails; fatal issue occurs either after two or three weeks (sometimes when the patient is apparently saved) or, more rarely, suddenly as a result of entrance of air into the veins. Radical operation has recently received enthusiastic advocacy.

Dentitio Difficilis

(DIFFICULT TEETHING)

As a rule, normal children get their teeth without any difficulty. They may show a slight indisposition in the form of fretfulness, disturbed sleep and slight loss of appetite. If care is being taken not to overfeed the baby during its teething period and the mouth is kept free from outside infection, there is rarely any need for special therapeutic measures. On the other hand, infants of low vitality and more especially those who had been suffering from gastroenteric disturbances or rachitis previous to the eruption of a tooth, teething, particularly if several teeth come at once, is very apt greatly to ag-

gravate the diseased conditions. But even in these children neglect in the general care of their health is responsible to a great extent for the serious consequences. Most people are so strongly imbued with the idea that teething is the sole cause of gastroenteritis, bronchitis, otitis, and what not, and that it must be so as a matter of course, that they complacently wait and watch for the teeth to protrude, and seek no medical aid to stay the ravages of the incidental ailments. It is usually in these cases that hyperpyrexia and convulsions are encountered, and that remedial measures have to be employed to facilitate teething, as it were.

Of course there are infants (see "Spasmophilia," p. 668) who will get convulsions, high fever, etc., on the most trifling provocation, and hence teething also is contributing its share in this direction, but all these extraordinary manifestations are no doubt exceptional.

The main points, therefore, are to reduce the food, to keep the child outdoors, and to avoid so-called "soothing syrups," which almost invariably contain opiates or similar stupefiers that depress the infant's vitality.

When the gum is very much swollen and the tooth visible directly under the mucous membrane, brisk friction (with rough end of sterile teaspoon) or even lancing of the gum does no harm and may relieve some reflex nervous symptoms.

DISEASES OF THE SALIVARY GLANDS

Salivation

Increased salivary secretion is almost physiologic during first dentition, and is the result of increased blood supply to the oral mucous membrane. Pathologically it is observed in stomatitis, cretins and other mentally deficient children; in helminthiasis and mercurial intoxication. Occasionally it is met with in apparently healthy children long after first dentition; and in the absence of any discernible cause it is attributed to a neurosis. In view of the harmlessness of the condition *per se*, no special treatment is indicated except protection of the chin and chest against the irritating effect of the constantly dribbling saliva, and removal of the causes wherever found.

Ranula

Retention cysts, congenital or acquired, are not rarely observed in children, and are the result of obstruction of the salivary ducts. Most frequently a globular, usually unilateral, tense, cystic swelling is found on the floor of the oral cavity, sometimes close to the frenulum.

This tumor which is designated *ranula,* varies in size from a pea to a pigeon's egg and contains a thin or viscid fluid. If large in size, the tumor interferes with suckling, swallowing and breathing, and calls for its incision and cauterization, or complete excision.

Ranula is not to be confounded with the peculiar *sublingual growth* (Riga's or Fede's disease) quite frequently observed in Italy* among nurslings. This neoplasm is usually situated at the insertion of the frenum linguæ, attains almost the size of a five-cent piece, and shows a tendency to return unless completely extirpated.

Secondary Parotitis

This form of inflammation of the parotid gland may occur in connection with acute infectious diseases. It differs from epidemic mumps (*q. v.*) in being, as a rule, unilateral. It heals spontaneously within a few days, or ends in suppuration, in the latter event requiring operative interference.

DISEASES OF THE TONGUE

Glossitis

Aside from the divers pathologic conditions of the tongue ordinarily met with in connection with stomatitis, tonsillitis, pharyngitis, exanthematous affections, etc., the tongue is subject to the following peculiar diseases:

1. **Glossitis Marginalis Erythematosa.**—The inflammation is usually limited to the edges of the tongue which are red and partially denuded of epithelium. It is observed in artificially fed infants, and is probably the result of mechanical irritation from the act of sucking, and more particularly from the constant use of the "pacifier."

The treatment is the same as for mild stomatitis.

2. **Glossitis Areata Exfoliativa (Annulus Migrans, Ringworm of the Tongue, Lingua Geographica).**—As a rule, it begins with a brownish thickening at the margin of the tongue, and, by gradual spreading, forms irregular, circumscribed lines, resembling, as the name indicates, a geographical map. Now and then part of the thickened epithelium is thrust off, but new places are soon involved, and in this manner the affection may go on for years, without, however, giving rise to ulceration of the tongue or any constitutional symptoms. It is not, as was frequently supposed, a sign of syphilis.

The treatment consists of cleanliness and occasional painting with a strong solution of chromic acid. (See "Stomatitis.")

---

*Only a few such cases have thus far been observed in this country.

DISEASES OF THE ESOPHAGUS

Esophagitis

Primary inflammation of the esophagus is comparatively rare in children, since the principal cause of the disease in the adult, *i. e.*,

Fig. 57.—Penny in esophagus of an infant readily extracted under the guidance of the roentgen ray.

corroding of the esophagus by caustic poisons taken with suicidal intent, is of exceptional occurrence. However, it is occasionally met with in connection with accidental injuries, such as impaction of foreign bodies, unintentional swallowing of caustics, etc., or scalding by hot

fluids. The accompanying symptoms vary with the extent of the injury. They consist chiefly of dysphagia, tendency to vomit, and expectoration of bloody, membranous masses. In severe cases, if the patient at all survives from the immediate effects of the injury (frequently fatal collapse), the esophagitis runs a very protracted course and produces secondary esophageal strictures (*q. v.*).

Secondary esophagitis occurs as an extension of inflammatory, especially diphtheritic, processes of the mucous membrane of the mouth and pharynx.

Treatment.—Antidotes in cases due to corrosives, morphine hypodermically for the relief of pain and shock, ice collar to the neck and ice by mouth to subdue the inflammation, and stimulants whenever indicated.

Stenosis Esophagi.—Esophageal strictures may be congenital (q. v.) or acquired, the latter being the result of esophagitis (q. v.). Depending upon the severity of the injury the stricture may advance up to total atresia. In children the stenosis is most frequently situated in the upper third of the esophagus, and may occasionally be detected by esophagoscopy. Otherwise the diagnosis is established by introduction into the esophagus of an elastic catheter or whalebone provided with a small olive-shaped steel tip. For this purpose the patient is placed in a sitting posture with the head extended slightly backward. The oiled instrument is guided with the first two fingers over the dorsum linguæ and the epiglottis into the esophagus.

In acquired stenosis the symptoms usually appear about two weeks after the injury and consist chiefly of difficult deglutition and gradual loss of weight. In cases of stenosis due to compression of the esophagus by diseased neighboring organs or tumors the symptoms are, of course, more gradual in their development and more intricate in nature agreeing with the primary cause.

Treatment.—Partial stenoses often yield to dilatation by means of bougies, provided the dilatation is continued two or three times a week for at least six months. The bougie is left in place for from five to thirty minutes. Occasional introduction of the bougie after apparent cure will prevent recurrences. Great care and patience are required to prevent perforation. Gavage and nutrient enemata are used if necessary. In severe and recurrent strictures operative interference (esophagotomy or gastrotomy) are in order. Good results are claimed from the use of thiosinamine: five drops of a 10 to 15 per cent glycerinated watery solution may be injected hypodermically twice a week in addition to the dilatation previously spoken of. Thiosinamine may also be given by mouth ($\frac{1}{2}$ gr. t. i. d.) and applied locally.

DISEASES OF THE STOMACH AND INTESTINES

General Etiology

With the recent advances in bacteriology and physiologic chemistry and corresponding improvements in sanitation and infant feeding, cow's milk no longer holds the record of *"Wuergengel"* (destroying angel) of the poor innocent babes. Indeed, a case of gastroenteritis is seldom met with which is not primarily traceable to some gross error of diet entirely independent of the cow's milk feeding. The sooner the physician will appreciate that fresh, unpolluted, properly modified (as to quality and quantity), well kept, and regularly administered cow's milk is not inimical (except, of course, in the comparatively rare cases of so-called "cow's milk idiosyncrasy" from birth) to good health and perfect development of the child, the better will he be prepared to reveal the etiologic factors of the gastrointestinal disturbance and combat them!

On the other hand, cow's milk, especially in the hot season of the year, whether contaminated at the dairy or at the filthy shop of the remorseless vendor, may form, like water, an excellent vehicle for the dissemination of pathogenic bacteria, and for the spreading of infectious gastroenteric affections.

Whatever the vehicle of transmission,—be it decomposed milk, fruit, vegetables, or meats; infected water, feeding bottles or nipples, cups or spoons, toys or fingers; infectious discharges from the mouth or nasopharynx, etc.,—careful investigation has established the fact that most, if not all, acute gastrointestinal diseases are primarily or secondarily due to microbic invasion of the alimentary canal, the severity of the affection more or less corresponding to the pathogenicity of the invading microorganisms.

The bacteria responsible for the production of gastrointestinal diseases are very numerous. Streptococci, the *B*. coli communis, *B*. dysenteriæ liquefaciens, (Shiga, Kruse and Flexner) staphylococci, *B*. influenzæ, *B*. pyocyaneus, *B*. proteus, among many others, contribute their share as etiologic factors. The determination of the specific germ of each type of gastrointestinal disease, however, is still a matter of experimental research and subject to great diversity of opinion.

Gastroenteric disorders in breast-fed babies may occur, in addition to errors of diet and exposure to infection—less frequent causes than in hand-fed babies—as a result of disturbance of the quality of the breast milk by disease, fright, grief, privation, pregnancy, and like influences on the part of the mother, or the wet nurse.

Finally, even in most carefully fed infants, gastrointestinal disorders are occasionally encountered where the alimentary canal is functionally or anatomically defective from birth (*e. g.*, pylorus stenosis), or where the infant is suffering from diseases of the other organs of the body, or is indisposed from the effects of functional or organic alterations associated with normal bodily development (*e. g.*, dentitio difficilis).

Stenosis Pylori Congenita

(PYLOROSPASM)

Stenosis of the pylorus may be complete or partial.

Complete atresia is extremely rare and invariably fatal from complete starvation within a few days after birth—sometimes before the diagnosis can be established.

Partial stenosis of the pylorus, on the other hand, is a comparatively frequent affection which not rarely terminates in recovery, either spontaneously or through medical and surgical treatment. It is distinguishable in two forms: True and false.

1. True or hypertrophic stenosis is invariably due to a congenital narrowing of the lumen of the pylorus and is associated with more or less *primary* hypertrophy of the pyloric ring and *secondary* dilatation of the stomach.

2. False or spastic pyloric stenosis (pylorospasm) is the result of congenital faulty innervation of the stomach, or of acquired digestive and nervous disturbances. It is free from primary hypertrophy of the pyloric ring. Sooner or later *secondary* hypertrophy of the muscular and mucous coats of the stomach occurs in consequence of the increased force required by continued muscular contraction of the stomach to propel the ingesta. At a later stage of the disease the stomach walls lose their tonicity and dilatation is the usual consequence.

The clinical picture of the disease is very typical. The apparently fully developed infant at birth, after a period of wellbeing of from a half to three weeks or even longer, begins to vomit sometimes after each feeding or after several feedings. The vomiting rapidly becomes very violent in character, and the contents of the stomach, which appear greater (*ischochymia*—retention of digested food) than the child could have taken in one feeding, consists of a hyperacid* mixture of mucus, digested and undigested food, free from bile, and is explosively ejected (projectile vomiting). As an immediate result of the vomiting, the intestinal tract remains empty; hence, absolute constipation, (but in fact only pseudoconstipation) or only occasional evacuation of a small quan-

---

*In two cases under our observation there was total achylia gastrica.

tity of brown, bile-stained, foul-smelling fluid. The urine is scanty and concentrated. The infant acts very hungry, voraciously swallows a few mouthfuls of food but being seized by sudden spas-
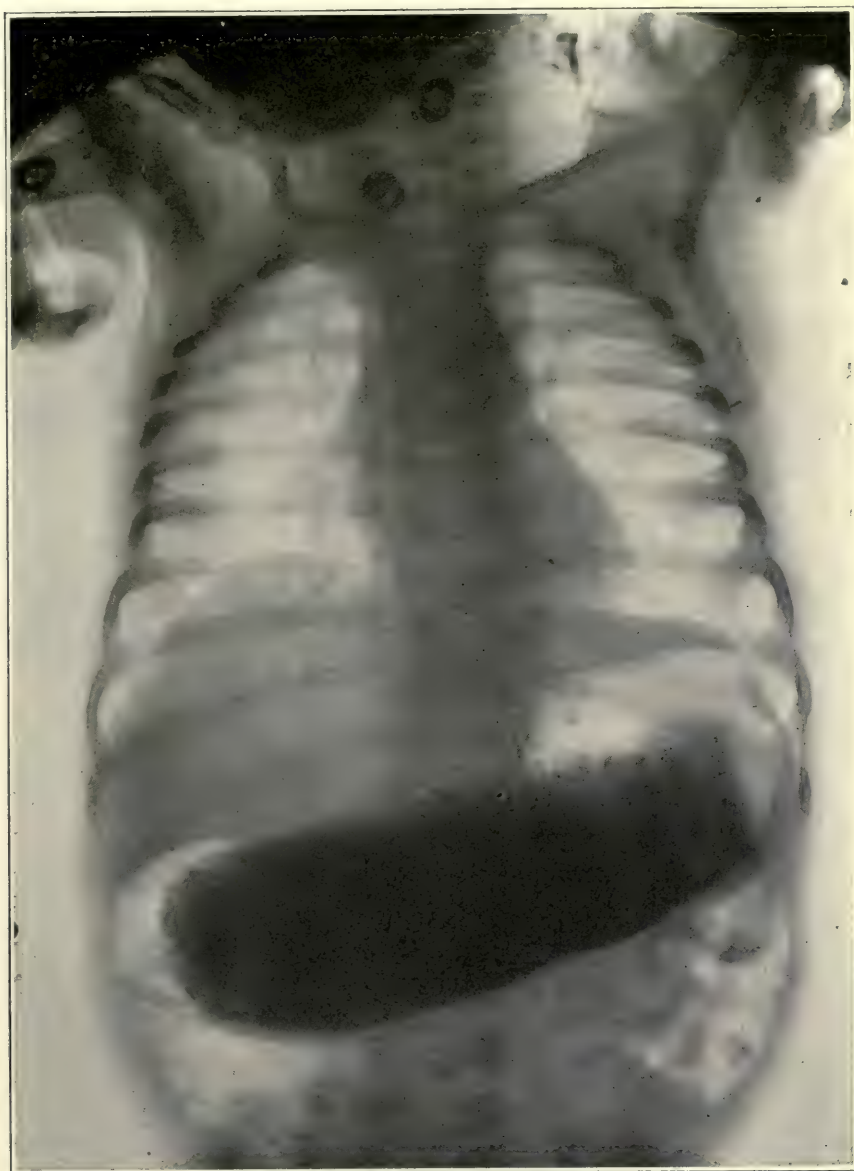
Fig. 58.—Pylorus stenosis in a boy three months old under observation of the author through the kindness of Dr. J. L. Rubinstein. Note almost complete closure of pylorus after bismuth test. Patient recovered fully after operation.

modic pain, it drops bottle or breast, only to grasp it again after some relief is obtained. The abdomen is sunken, while the epigastrium is distended, and here and there are visible peristaltic movements (*hyper-kinesis*) of the stomach, from left to right. The peristalsis can be seen and felt only after the infant has become greatly emaciated. Occasionally the peristaltic movement is *reversed, i. e.*, from right to left. The peristaltic stomach wave is best obtained by washing the stomach and allowing 2 ounces of water to remain, or giving 2 ounces of food. If the patient is given a pacifier and is then placed on his back with the light favorable for observation, the wave phenomenon will shortly appear (Kerley). In most cases a small tumor—the hypertrophied pylorus—is palpable at the pyloric end of the stomach a little above and to the right of the umbilicus, or lower down after the stomach has become very much dilated.

In early stages of *pylorospasm* the symptoms are less pronounced, vomiting is less frequent, and the stools contain some curds and hardened feces, but otherwise cannot be easily distinguished from true pyloric stenosis except by Roentgen-ray examination. In pyloric obstruction, bismuth subcarbonate (administered through a tube) will fail to enter the intestines, or do so only after a period of twenty-four hours and in very minute quantities. Less reliable is the charcoal test. This consists in the administration by the stomach tube of 10 grains of charcoal in 2 ounces of water and examining the contents of the stomach for the charcoal twenty-four hours later.

The course of the affection varies with the degree of contracture. In the majority of instances the *true* form of the disease, if not operated upon early, terminates fatally in from four weeks to four months, with symptoms of inanition, acidosis, and collapse, or pneumonia. Occasionally, however, a change for the better occurs and slow recovery follows. This is particularly apt to take place in spastic pyloric stenosis, especially if early and properly treated. With these facts in view, it is extremely difficult to decide when and whether surgical intervention is indicated. The profession is greatly divided on this question. The statistics adduced for and against an operation seem to favor both contentions. The surgical "cures" do not always assure us of their permanency. A little patient of mine, nine weeks old, recently operated upon, did well for six days, but died two days later from the effects of a minute gastrointestinal fistula. Two of my patients were operated upon apparently successfully, but died suddenly a few weeks later. On the other hand, who can vouch for the permanency (remissions are not rare!) of the medical "cures," and for the correctness of the diagnosis in

such cases! H. Lowenberg offers the following very valuable observations, as to the type of cases demanding operative treatment.

NONSURGICAL	SURGICAL
1. Weight curve resembles curve of continued fever with slight remissions and elevations. At the end of a week it is stationary, or but slight loss or gain is recorded.	1. Weight curve resembles the crisis of a pneumonia. End of a week records a loss of 8 to 10 ounces or more.
2. General strength is not materially reduced at end of this time.	2. General strength fails rapidly.
3. Bowels are constipated but movements are of fair size and contain curds or digested milk.	3. Constipation absolute or nearly so. Movements are ordinarily of bile-stained mucus, sometimes very small amounts of milk feces.
4. Recovery of considerable quantity of charcoal in rectal discharges, although its passage is delayed.	4. Nonrecovery of charcoal, or very little at end of thirty-six to forty-eight hours and continuing for many days.
5. Nonrecovery, or recovery of but little charcoal in the stomach washings twenty-four hours later.	5. Recovery of considerable quantity of charcoal in the stomach washings twenty-four hours or more after administration.
6. X-ray examination reveals more or less bismuth in the small and large intestines.	6. X-ray pictures taken in series for a period of twenty-four hours show retention of bismuth in the stomach, and not any or only traces in the small and large intestines. Bismuth shadow has a ''comet''-like appearance.
7. Severity of vomiting is intermittent and often yields to gastric lavage.	7. Constant, and not influenced by gastric lavage.
8. Constantly palpable, except before mittently so.	8. Constantly palpable, except before emaciation occurs.

An operation, if indicated, should not be delayed until the child is at death's door. The choice between divulsion (Loreta), pyloroplasty, gastroenterostomy, posterior gastrojejunostomy and the Rammstedt operation (splitting of the pylorus longitudinally down to but not through the mucosa), depends upon the pathologic condition of each individual case, and the judgment of the surgeon.

In reviewing one hundred and seventy-five cases of plyoric stenosis in which the Fredet-Rammstedt operation was performed, W. A. Downes[*] offers the following suggestions:

[*]Jour. Am. Med. Assn., July 24, 1920.

1. If the patient is observed from the onset of symptoms, medical treatment may be tried for a period of not longer than ten days, provided the weight loss does not exceed 20 per cent during this time. If, at the end of this period, the child does not show definite improvement, operative interference is indicated. Any patient, continued under medical care, and suffering a relapse should be operated on at once.

2. All cases in which there is a history of a period of ten days or longer in which the data as to previous weight are lacking—and in which the patient is not in very good condition—should immediately be classed as surgical.

3. The mortality among patients coming to operation within four weeks from the onset of symptoms is less than 8 per cent.

4. The results following the Fredet-Rammstedt operation are permanent and the cure complete.

The Rammstedt operation is highly recommended by E. Feer. Dufour and Fredet have collected 36 cases operated by the Rammstedt method with 9 deaths. Kerley reports 26 cases with 4 deaths, and believes that operation by the Rammstedt method will insure a mortality of only 5 per cent in patients who have not vomited more than two weeks, provided, of course that adequate after-care, which includes the use of breast milk, is supplied. He recommends the following *postoperative management*, as evolved by Holt and W. L. Downes. The infant is wrapped in a warm blanket before leaving the operating room and when in bed is surrounded by hot water bottles *outside* the blanket. For an hour or two following the operation, the head of the bed is kept lowered to prevent aspiration of mucus into the larynx. This is absolutely necessary while the infant is still under the influence of the anesthetic. When nourishment is commenced, the head of the bed is raised to a level position. Ten or twelve hours later the patient is placed in a semierect position, which tends to prevent regurgitation of food and permits the more easy escape of gas.

As soon as the patient is placed in bed, a hypodermoclysis of 120 c.c. of physiologic sodium chloride solution is given, and if the condition is poor, a hypodermic of 5 minims of epinephrin, 1:1,000, is given and repeated in from four to five hours. Dilute whiskey, 5 minims every three hours for the first five or six days, has proved of great value. Transfusion in a few cases of collapse has been of material benefit, from 80 to 120 c.c. of blood from either parent being given preferably into the median basilic vein.

One and one-half hours after operation, provided the patient has sufficiently recovered from the anesthetic, 10 c.c. of water are given, and one and one-half hours later, 4 c.c. of barley water and 4 c.c. of breast

milk. Two hours later, 8 c.c. of breast milk and 4 c.c of barley water are given. Breast milk is then given every three hours, alternated with water, and gradually increased in amount so that at the end of forty-eight hours about 30 c.c. are given at a feeding, with 4 c.c. of barley water. The barley water is then discontinued, and on each successive day the amount of milk permitted is increased 5 c.c. at a feeding, so that by the eighth day following the operation the patient is having 60 c.c. every three hours. On the third day the intervals of feeding at night are lengthened to four hours, so that seven feedings are given instead of eight. By the time the baby is taking 60 c.c. of breast milk at each feeding, he may be put to the breast once. The baby is weighed before nursing and at intervals of three minutes until he has nursed 60 c.c. from the breast. The following day three nursings are allowed, so that by the eleventh or twelfth day the patient is nursing entirely and is able to leave the hospital. Measurement of food during the nursing must be kept up one week later by carefully weighing the baby both before and after nursing. For one month or longer a wet nurse is advisable if the mother is not able to nurse her child.

In well-nourished infants, a sponge bath is given daily until the wound is completely healed. In emaciated children, an oil rub is preferable until such time as the tendency to subnormal temperature has passed.

In cases of vomiting due to accumulation of gas in the stomach, the child should be raised to an upright position after feeding. If this does not suffice, a soft rubber catheter may be passed into the stomach before each feeding. If still persistent, gastric lavage may be employed. One teaspoonful of castor oil is usually given twenty-four hours after operation if there have been no stools. There should be from two to three stools a day. If they are more frequent, protein milk may be substituted for three or four breast feedings.

The wound is covered with a narrow fold of sterile gauze held in place by adhesive strips. It is not disturbed for four or five days, unless some indication arises. The stitches are removed on the ninth or tenth day.

The *nonsurgical* treatment of congenital pyloric stenosis must be carried out systematically and faithfully. Whenever possible, the infant should be fed on woman's milk, (after removal of fat), preferably with a spoon or tube,* in order to gauge the amount of food consumed, and possibly retained, by the infant and also to avoid contractions of the stomach by the act of sucking. The amount of feeding should not exceed

---

*P. Hertz (Ugeskr. f. Laeger, June 13, 1918) recommends duodenal feeding by means of a Nélaton catheter, No. 18 or 19, giving as much as 2 to 3 ounces at each feeding.

one ounce, but may be given every hour or two, so as to sustain the child's vitality. Modified or predigested milk may be administered instead of woman's milk if the latter is not readily obtainable. In view of the fact that almost two-thirds of the cases of pyloric stenosis thus far reported were breast-fed babies, one is tempted to recommend fat-free cow's milk feeding as a therapeutic or, at least, as a prophylactic measure against pyloric stenosis. Indeed, following the temptation in 2 of my own cases, I was—perhaps accidentally—rewarded with happy results. May I venture to suggest that the large curd of cow's milk tends mechanically to dilate the contracted pyloric orifice? and, furthermore, that the fat breast-milk is possibly a cause of pylorospasm?

Reduction in the frequency of the attacks of vomiting and in the amount ejected forms the first and best indication of improvement in the condition. Next to careful feeding, systematic washing of the child's stomach serves as the sheet-anchor in the therapeusis of congenital pyloric stenosis. It should be practiced at least twice a day with plain, cool (70° to 80° F.) water, occasionally adding a small amount of bicarbonate of soda to neutralize the hyperacidity of the stomach. The washing should be continued until the water returns clear. The effects of the lavage are the removal of decomposing substances from the stomach, arrest of fermentation and the allaying of pain and spasm. For the latter purposes, prolonged warm baths and hot compresses to the epigastric region are also very useful. To counteract the excessive loss of fluids, a daily enteroclysis or hypodermoclysis is of advantage. Internal medication is of little value, except anodynes for the relief of pain and spasm. For this purpose minute doses of codeine with or without belladonna may be administered in the form of suppositories.

S. V. Haas (*Am. Jour. Dis. Child.*, May, 1918) prefers atropine owing to its paralyzing effect on the vagus nerve endings. In the course of twenty-four hours he administers from 1/50 to 1/25 of a grain with an extreme of 1/16 of a grain, divided among the whole day's feedings.

Skillful nursing, privately or in a hospital, should be insisted upon.

I. Acute Gastroenteritis

(INDIGESTION, DYSPEPSIA)

Classification

In accord with the aforementioned general etiology (see p. 241) gastrointestinal disease in infants and young children may be classified as follows:

1. *Dyspepsia ex alimentatione,* or faulty assimilation of the food, as a result of:

A. Overfeeding or too frequent feeding in general, or

B. Overfeeding with a milk mixture containing too much of:
    a. Fat, b. Carbohydrates, c. Proteins.

2. *Dyspepsia ex infectione*, due to:
    A. Direct infection of the intestinal tract, (enteral).
    B. Indirect infection (parenteral), *i.e.* secondarily to other diseases.

3. *Dyspepsia ex constitutione*, in consequence of:
    A. Congenital deficiencies
        a. Organic, *e. g.*, pylorus stenosis, megacolon.
        b. Functional or constitutional dyscrasia, *e. g.*, exudative diathesis.

As the other varieties of gastrointestinal affections are fully described in other parts of the book (see pp. 521, 522) we will here limit our discussion to the infectious gastroenteric affections.

Occasional vomiting and diarrhea, occurring as a result of unusual overloading of the stomach, too hasty feeding, the partaking of indigestible articles of food (raw, unripe fruit, peels and parings), or foreign bodies, exposure to sudden atmospheric changes and undue excitement, etc., are not rarely observed in otherwise apparently healthy, well-nourished children, and if of brief duration, are of no special clinical significance. These attacks may even be accompanied by fever, mild cerebral irritation, colic, etc., and yet remain outside the domain of pathology, or represent an affection which is generally spoken of as simple indigestion or the *first stage* of gastroenteritis. By avoiding further transgressions of the ordinary dietary and hygienic rules, and by removing the causal obnoxious influences, recovery is usually prompt and permanent.

If, however, the vomiting and diarrhea persist or recur at frequent intervals; if the child loses its appetite and some of its weight; if its tongue becomes heavily coated, its abdomen greatly distended and its general health more or less seriously impaired; if the infant suffers from severe abdominal pain after each feeding and vomits part of the food consumed and some mucus and bile; finally, if the stools rapidly increase in number and consist of masses of undigested food, of bad color and offensive odor, a symptom-complex develops which represents the *second stage* of gastroenteritis and is generally described as gastrointestinal catarrh or dyspepsia.

Ordinarily these manifestations set in insidiously, and, if not promptly arrested, grow worse gradually, arousing little if any anxiety on the part of those in charge of the baby, or are lost sight of, sometimes because of coincident "teething" (with the laity the presumptive cause of all ills), until there is a sudden aggravation of the condition—supervention of the *third stage* of the disease.

In this stage, gastroenteritis assumes a very acute course. It is manifested by violent vomiting, excessive thirst; frequent, thin watery, brownish, greenish, and later colorless or blood-stained stools. The vomitus is acid in reaction, bile stained, and offensive in odor. The bowel movements vary between ten to fifteen in twenty-four hours, are preceded and followed by griping pain and tenesmus. The child is very restless, feverish, sleepless, and, with the symptoms persisting a few days, rapidly loses in weight, and sinks into a state of collapse, followed by convulsions, coma and death. More favorable cases may improve under energetic treatment (see "Cholera Infantum," p. 251), or linger for weeks or months, frequently suffering from intense exacerbations of the attacks, and, finally, either recover after tedious convalescence or die from inanition or complications.

Cholera Infantum

(Summer Complaint)

Closely allied to the gastroenterocolitis just described (though possibly differing as to the exciting microorganism—probably the dysentery bacillus, but also the gas bacillus or streptococcus), and probably representing only a severer, "fulminating" form of the same disease, is the so-called infantile "summer complaint" or *cholera nostras s. infantum*. It usually rages in epidemic form during the hot summer months, especially among bottle-fed infants and those exposed to bad hygienic conditions, but occurs sporadically also at other seasons of the year. As with other contagious and infectious diseases, previous ill health serves as an active and favorable predisposing cause also in this destructive affection, the acute and grave symptoms ordinarily supervening upon a latent period of indisposition of variable duration.

The attack ushers in suddenly with vomiting, diarrhea and prostration. The vomiting is more or less projectile in character and occurs especially immediately after drinking. The evacuations range between fifteen to thirty, or more, in twenty-four hours, are at first fecal in consistency and odor, but soon turn very watery, serous, light yellow or greenish in color, and occasionally are mixed with blood-streaked mucus. The abdomen is often trough-shaped and but slightly sensitive to pressure. The thirst is intense; the tongue dry, brown or black and cracked, irrespective of the degree of temperature, which is rarely very high. Owing to the excessive loss of fluids, the urine is very scanty and often contains a moderate amount of albumin.

As the disease progresses the child perceptibly loses in weight, from hour to hour; its face is pinched, its fontanelles, temples and eyes

PLATE IV

THE GREEN, ACID STOOL OF DYSPEPSIA

Typical whitish curds of soap scattered throughout its substance. Apart from the soap curds, the stool is so watery that it forms a mere stain on the napkin. A little greenish mucus is also present.

(Courtesy Dr. Hector Charles Cameron.)

are deeply sunken; its extremities are cool and blue; the heart beat and respiration barely audible—in short, the child is in a state of profound collapse. Apathy, somnolence, convulsions and death then follow in rapid succession; the younger the child, the earlier, as a rule, the fatal termination. The latter is sometimes preceded by a state of *hydrocephaloid*—a condition variously ascribed to cerebral anemia or hyperemia, edema of the meninges and uremia, and presenting the following symptom-complex: *First stage*, fever, restlessness, jactitations, and insomnia, flushed face, strong and bounding pulse; *second stage*, subnormal temperature, cold extremities, feeble, irregular pulse and respiration, apathy, sopor and coma.

The disease having reached this grave stage, it offers a very bad prognosis; few children manage to survive so violent an attack. Some of the few who do, are apt to succumb later to complicating nephritis, pneumonia, cerebral sinus thrombosis, peritonitis and the like.

Convalescence is very tedious even in the absence of complications, and a great many children remain decrepit for life; chronic otitis media, xerosis of the cornea and panophthalmia often adding to their share of misery.

With such sad prospects in view after the gastrointestinal affection is fully established, the urgency of early and energetic prophylaxis and treatment can readily be appreciated.

Treatment.—To prevent the graver forms of gastroenterocolitis we must promptly remove the causes and effects of the mildest symptoms of the disease. Attention to every detail of rational feeding and personal hygiene and strictest cleanliness of the child's living rooms, feeding utensils, wearing apparel, and of all other things coming in direct contact with the patient are the surest means of prevention. As in the majority of instances, the pathogenic bacteria enter the infantile alimentary tract with infected milk or water, these should, especially in the summer months, be sterilized or even boiled, regardless of the temporary arrest of gain in weight that is concomitant with such feeding—a puny baby on the lap, rather than a fat one in the grave! Weaning of the baby and other innovations during the hot summer months should be avoided. Lengthy voyages, exacting prolonged disturbance of rest, sleep, and improper feeding should be interdicted. On the other hand, a sojourn in the country (inland, mountains, or seashore) should be encouraged. Last but not least in importance as a prophylactic measure is the practice of whole or partial breast feeding of infants under one year of age, unless countermanded by definite contraindications.

The active treatment should begin, as already suggested, with the earliest inception of the gastrointestinal disorder. Regulation of diet is our most efficient therapeutic measure, and is almost invariably attended by improvement in the child's condition, if it is begun with a few hours' starvation of the patient and prompt cleansing of the alimentary tract of its obnoxious contents. Feeding, breast or bottle, should at once be suspended until such time as exigencies for resumption of feeding shall demand. In the meantime, especially in the absence of strong contraindications, such as violent vomiting, the infant should receive small quantities of hot or cold pure water or a light infusion of black tea, sweetened with saccharin. Recurrent vomiting calls for prompt attention, especially because of its fearfully exhausting effects, but also, because it greatly hinders in the administration of suitable medication. Ordinarily vomiting can be controlled by "ice-sand," minute doses of calomel with large doses (gr. x) of bicarbonate of soda; bismuth and cerium oxalate; tincture of iodine (in 1/30 of a drop doses, to be repeated every hour or two) ; and, if all else fail, lavage. In hospital practice the order of these therapeutic suggestions is usually reversed, i. e., lavage is usually resorted to first, and, as a rule, with immediate relief to the patient. In private practice, however, one often meets with objections on the part of parents, and hence is obliged primarily to "medicate." Lavage should be supplemented by enteroclysis and, with the vomiting checked, also by a small dose of castor oil.

This mode of treatment generally suffices to arrest gastrointestinal affections of moderate severity. Where the diarrhea persists, we are often called upon to administer an astringent mixture like the following :

> ℞ Bismuthi subcarbonatis,
> Mist. cretæ comp.,
> Syr. rhei aromat.,
> Glycerin.,
> Aq. menthæ pip. ....āā ʒij | 8.00
> Aq. destil. ....q. s. ad f℥ij. | 60.00
> M.
> S.—One teaspoonful every two hours for a child one year old.

The camphorated tincture of opium may be added for the relief of pain. After complete cessation of vomiting, we may resume feeding, first with small quantities of toast- or barley-water or dextrinized gruel (cereo), and several hours later, diluted protein milk (1 ounce of the milk with an equal or a larger quantity of water, and later cereal water). After the diarrhea has been arrested, the feeding with

breast milk or modified cow's milk in small and gradually larger quantities, may be resumed.

In fulminating attacks of gastroenterocolitis, where the bacterial toxins so violently overwhelm the infantile organism and produce intense shock, the treatment must be very prompt and more heroic. In the *initial*, febrile stage, after a single but thorough irrigation of the stomach and bowels, the little patient is given 1/50 of a grain of morphine and 1/500 of a grain of atropine hypodermically, is wrapped in warm blankets and sent outdoors—wherever a good breath of air is obtainable —preferably to the seashore. After responding favorably, the treatment is followed up in the manner previously outlined for less severe cases.

In the *algid* stage, where the child is at death's door—wasted, cold, blue, rigid and lifeless, in short in profound collapse—powerful stimulation is in order. Thus, a hot bath with brisk rubbing of the body; a hot (110° F.) high enema (injected slowly so as to be retained), hot water by mouth, hypodermic administration of sterile camphorated oil (8 drops of a 15 per cent solution), strychnine (gr. 1/60 to 1/30), caffeine sodium benzoate (1 grain), or whiskey (10 drops), hypodermoclysis (1 to 6 ounces of a 0.9 per cent hot sterile salt solution), and injection of normal saline in the peritoneal cavity or longitudinal sinus (see p. 209). As the patient improves a milder course of treatment is, of course, resorted to. The physician should not be deceived, however, by those apparent improvements, as they not rarely precede fatal termination.

II. Subacute and Chronic Gastroenterocolitis

Exhausted by the paralyzing action of the virulent bacterial toxins; wasted and weakened from the excessive loss of body fluids and the strict starvation diet enforced during the acute course of the disease, the little patient rarely, if ever, emerges in a state of health capable of exercising its digestive organs to their normal capacity. On the contrary, convalescence usually proceeds at a very slow pace, and is frequently interrupted by milder exhibitions of gastrointestinal indigestion, which, if not promptly yielding to energetic treatment, eventually lead to chronic involvement of the alimentary tract.

The mucosa of the stomach and bowels, especially of the ileum and colon, undergoes gradual thickening, and often ulceration. The mesenteric glands are more or less enlarged, and on cross-section are partly red and partly yellowish gray in color and sometimes caseated. In very protracted cases the mucosa and its follicles are atrophied, and the lungs, liver and heart are in a state of inflammation and degeneration.

The bowel movements continue to be frequent (four or five times in twenty-four hours). The stools are thinner than normal, are mixed with particles of undigested food, mucus, and blood. The abdomen is flat, sometimes deeply sunken, and through its thin and wasted wall one can readily palpate the greatly enlarged, "ropy," mesenteric glands. The child's appetite is capricious, often very good, and contrasts strongly with the persistent loss of weight. The tongue is coated and flabby, its edges are red and indented by the teeth or

Fig. 59.—Chronic gastroenteritis in an infant ten weeks old. (See Fig. 56.)

gums, and here and there covered by an aphthous deposit. Slight indiscretions in the dietary are promptly followed by vomiting and diarrhea. Chemical examination of the contents of the stomach discloses marked diminution of hydrochloric acid.

The course of chronic gastroenteritis varies in individual cases. Some infants, especially those in whom the chronic affection followed upon the acute form, who remained free from grave complications and retained some vitality, often unexpectedly show marked improvement

with the setting in of cooler weather, and regain their health fully within a few weeks.

In another group of cases recovery is less rapid. Improvement alternates with aggravation of the condition, but, finally, the infant extricates itself barely alive, with a load of sequelæ (*e. g.*, rachitis) which keep it in a state of decrepitude for many months and even years thereafter.

In still another group of cases all therapeutic efforts utterly fail to effect a cure. The child's face has a pallid, earthy tint, and senile expression; the skin is dry and hangs in folds; the fontanelles and temples are depressed, and after a period of several weeks or months the infant finally succumbs either slowly with symptoms of cerebral anemia and heart failure or suddenly during an attack of eclampsia. The fatal termination is frequently enhanced by complicating pulmonary (passive- or bronchopneumonia) and renal (colicystitis, pyelitis, etc.) affections; skin (ecthyma, furunculosis), ear and mouth infections, or intercurrent acute communicable diseases (exanthemata).

At best the prognosis is very grave (30 per cent mortality), especially so in infants reared under bad hygienic conditions, in want and misery, and in those born with lowered vitality and congenital defects.

However, no effort should be spared to save an infant that is apparently hopelessly lost, for just in chronic gastroenteritis the unexpected sometimes happens—recovery takes place at a time when relief by death is prayed for.

Treatment.—The patient should be removed from insanitary surroundings and intrusted to the care of some one who will *obey* orders rather than use her own judgment and that of the many "good and experienced" neighbors. Be it remembered, that only too often change of nurse (with her gross negligence and stubborn interference) has saved many a hapless baby! Regulation of diet is most essential. No hard and fast rule, however, can be laid down in this direction. We must feel our way in every individual case. It is always a good plan in bottle-fed babies to begin treatment with discontinuance of the milk for a day or two and thorough cleansing of the alimentary tract by a laxative, lavage and enteroclysis. In the meantime the patient should be fed on thin barley water, acorn cocoa, a light infusion of black tea, albumin water, diluted protein milk, and perhaps, a small quantity of freshly boiled, fat-free chicken or mutton soup. As soon as the stools diminish in frequency and improve in consistency, we resume milk feeding in very high dilution. For a child, let us say of six months, one tablespoonful of fat-free milk to seven tablespoonfuls of barley or rice water,

to be given every three hours, may be prescribed, and directions given daily to increase the quantity of milk until the percentage of 1 to 2 has been reached; then gradually the total quantity at the last ratio (*i. e.*, 1 to 2) is augmented, until 6 ounces are obtained for each feeding. Should the milk mixture disagree, a weaker milk mixture is resorted to, or milk is again discontinued, falling back upon albumin milk with cereals, albumin water and tea. Some infants do well, at least for a time, on condensed milk and barley water; others, especially those suffering from the so-called "fat-diarrhea," improve rapidly on albumin milk, skimmed milk or whey, and still others (older ones), who cannot tolerate milk in any form, get along on toast and tea, acorn cocoa in water, mashed potato with beef juice or chicken soup, soft-boiled egg, ground rice custards and similar semisolid articles of food. In a great many instances "malt soup," prepared in accordance with the directions of Keller, acts admirably, both as a tissue builder and to check the protracted diarrhea. Last in line, but foremost in importance, is the fact that in young infants a complete cure of chronic gastroenteritis in bottle-fed infants is effected by a prompt change from bottle to breast feeding.

The medicinal treatment of chronic gastroenteritis is chiefly symptomatic. When vomiting persists, lavage (with warm boric acid solutions) should be practiced daily or every alternate day, and, if need be, continued for a few weeks. Digestion may be aided by means of pancreatin and diastase, and the appetite improved by small doses of tincture nux vomica and dilute hydrochloric acid and pepsin. The patient should be given daily a low intestinal irrigation, either with one quart of plain hot (110° F.) water, 2 per cent of bicarbonate of soda, or, where the lesion is localized principally in the lower bowel— as indicated by predominance of blood and mucus in the evacuations— with 1/10 per cent solution of nitrate of silver. Where the diarrhea persists notwithstanding progressive improvement in the general condition of the patient, the newer tannin preparations (*e. g.*, tannalbin, tannigen) are very serviceable. The tannates may be combined with some bismuth preparation(*e. g.*, subgallate of bismuth, 2 to 4 grains), to enhance the astringent effects, and small doses of Dover's powder (¼ grain every three hours) to arrest active peristalsis.

Change of air (seashore), strict cleanliness of the body, change of position and frequent picking up of the patient from its bed, and active stimulation (strychnine, cinchona, Tokay wine and champaign) are active preventives of serious complications.

Dysentery, Enterocolitis, Ileocolitis, (See page 412)

Acidosis (See p. 522.)

Proctitis

Inflammation of the rectum is usually secondary in character and not rarely associated with gastroenterocolitis, dysentery, oxyurides, and prolapsus recti, and less frequently with gonorrhea (vulvovaginitis, (*q. v.*) and diphtheria. Occasionally it is the result of trauma (*e. g.*, foreign body), and the effect of drastic cathartics.

The principal symptoms of this affection consist of tenesmus (sometimes also strangury), frequent discharge of blood, mucus, and pus, with little fecal matter, and more or less severe colic. Depending upon the primary cause of the disease, the discharges may contain different kinds of bacteria (*e. g.*, ameba, gonococcus, diphtheria bacillus; worms, etc.), a fact which should always be borne in mind before arriving at a diagnosis and resorting to treatment. Proctitis should not be confounded with rectal fistula, polypus or hemorrhoids, purpura hemorrhagica and intussusception. The treatment depends upon the underlying cause; in the main resembling that of dysentery (*q. v.*).

Colica Infantum, Gastralgia, Enteralgia, Neuralgia Enterica

Infantile colic is usually associated with a number of congenital (gastrointestinal stenosis, etc.) and acquired (gastrointestinal inflammations, etc.) diseases of the alimentary tract. Less frequently it is apparently free from organic underlying causes. This so-called "idiopathic" form of colic is a spasmodic affection of the intestinal musculature, the result of pathologic irritations which act by way of the peripheral cutaneous nerves or the sensory intestinal nerves. To avoid unnecessary repetition, it may briefly be stated that anything capable of producing gastrointestinal disturbance may form the cause also of the said pathologic irritations. This occurs especially in premature infants and in those whose digestive organs are not quite fully developed.

Some babies, breast or bottle fed, begin to suffer from colic soon after birth, and do what you will, maintain their "record" for several months,—until, with gradual growth, the digestive organs attain their normal functions. Such "colic-babies," if reared without immediate strict supervision of a capable nurse or physician are apt very soon to contract a severe gastrointestinal disorder from the effect of the experimental efforts, in feeding and medication, on the part of all who sympathize with the "innocent babe." This *habitual* colic, which is manifested by continued fretfulness, sleeplessness, and *pseudobulimia*

(instinctive, eager desire for warm drinks which temporarily relieve the pain), is to be distinguished from *acute* intestinal colic (colica flatulenta), which is sudden in development and rapid in disappearance, the latter depending upon the time required to get rid of the gas or stool. During a severe attack of acute colic the child's face is spasmodically drawn and bathed in perspiration. The patient refuses food, cries pitifully, and draws its legs upon the abdomen. The spasm sometimes extends to the other muscles of the body, leading to general convulsions, and exceptionally even to coma and fatal issue. Of course, in the majority of instances, the termination is favorable, especially under prompt and appropriate treatment.

Treatment.—In breast-fed infants attention to the health of the mother or wet-nurse—avoidance of excitement, regulation of the bowels, indulgence in outdoor exercise—and in both breast and artificially fed, prevention of constipation and overfeeding of the infant, more particularly with fat mixtures, are very efficient anticolic measures. Where repeated examination of the breast milk proves it to be too rich in fat or protein, the infant should be given a few teaspoonfuls of water or of some other diluent immediately before each nursing, and the length of time for each nursing proportionately reduced.

As long as the infant thrives, notwithstanding the colic, no very material changes in the feeding should be attempted, as too much experimenting often makes matters worse.

In habitual as well as flatulent colic, heat, either in the form of fomentations (a few drops of turpentine in a quart of warm water), gentle massage of the abdomen with warm oil, or warm drinks such as chamomile, fennel seed or peppermint tea, will be found to act well. In cases of acute colic this must be preceded by a warm water enema to aid in the expulsion of the gas or stool. Of drugs, the following preparations are worth trying:

Charcoal and magnesia, of each 1 or 2 grains one hour after feeding; mistura sodæ et menthæ, N. F., 5 to 10 drops every ten minutes until relieved; compound spirits of ether, sweet spirits of nitre, or camphorated tincture of opium in doses of from 2 to 5 drops, to be repeated two or three times. In purely nervous colic asafetida often acts magically. The lac asafetida (½ dram to 1 pint of warm water) should be gently administered by rectum. The ammoniated tincture of valeria (5 drops) and sodium bromide (2 grains) are often equally efficient. As to the treatment of convulsions, see p. 670.

Proper food, regular bowel movements, and fresh air are efficient prophylactic measures.

Infantile colic should not be confounded with intestinal intussus-ception, appendicitis, and biliary, renal (uric acid infarct!), or vesical calculi.

Chronic Constipation

Judging by the construction of the infantile intestines—their great length, the thinness and feebleness of their musculature, etc.—nature seems to have intended that infants as well as older children should be more or less constipated. Indeed, the popular belief that healthy children are usually constipated, is often corroborated by actual ob-servation. Not infrequently, however, obstinate constipation gives rise to a number of disagreeable symptoms (flatulence, anorexia, head-ache, restlessness, sometimes convulsions; anemia, toxemia, a tendency to renal irritation, possibly with colicystitis; proctitis, anal fissure, pro-lapse of the rectum, hemorrhoids, etc.) requiring active treatment, a task often difficult to cope with in view of the uncertainty of the etiologic factor of the underlying disease.

The causes of habitual constipation are very numerous. Aside from the cases resulting from gross abnormal anatomic relations or diseases, such as the different varieties of atresia intestini, recti, or ani; tumors; congenital dilatation with hypertrophy of the colon; hypertrophy of the valvulæ conniventes; hypertrophy of the so-called rectal valve; in-flammatory adhesions; congenital displacements—which will not be discussed here—constipation is ordinarily caused by faulty diet, atony of the bowels, and constitutional disturbances.

Faulty diet is responsible for a great many cases of constipation. This etiologic factor is frequently potent also in infants, when the woman's milk contains too much or too little of one or more of the constituents of milk, or when it is insufficient in quantity. In arti-ficially fed infants the cause of the constipation will probably be found in the excess of fat consumed. In some children constipation is due, on the one hand, to too early and persistent feeding with amylaceous food, and, on the other hand, to the consumption of food that does not stimulate peristalsis, such as an exclusive diet of milk, meat, eggs, etc., and no fruit, potatoes, bread, fresh vegetables, etc.

Atony of the intestines may be primary, congenital in nature, or secondary or acquired. The former variety can frequently be traced as an hereditary taint through several generations. Sometimes there is, in addition to the muscular insufficiency, also congenital weakness of the innervation of the intestines. The latter condition embraces also the form of atony usually associated with congenital diseases of the brain and spinal cord. Secondary or acquired intestinal atony is generally

the result of repeated attacks of temporary constipation, gastrointestinal indigestion with fermentation, enterospasm, arrest of peristalsis due to reflex irritation of the inhibitory nerves of the intestines, acute inflammatory processes of the intestinal canal with consecutive atrophy of the intestinal coats, constriction of the lumen of the bowels by temporary displacements (enteroptosis, hernia, etc.), habitual suppression of defecation or attention to it at irregular hours, enemas with large quantities of fluids, etc. All these etiologic factors produce intestinal atony by directly or indirectly distending the lumen of the bowels and depriving the intestinal musculature of its resilience and tonicity.

In different chronic diseases associated with general debility (e. g., rachitis) and loss of flesh; in diseases of the nervous system, such as infantile paralysis, myelitis, meningitis, etc., the sluggishness of the bowels forms merely a symptom of the principal disease. Habitual constipation is often met with in diseases of the heart, profound anemia, etc., as a result of venous stasis of the abdominal organs. To the same cause is attributable also the constipation occurring in children who, through deformity or when otherwise afflicted, are incapacitated to enjoy a sufficient amount of bodily exercise.

The *treatment* of obstinate constipation in infancy and childhood resolves itself, first, in arresting the causes instrumental in the production of the disease; secondly, in the removal of the damage done during the continuance of the constipation—not quite as easy a task as some authors wish us to believe. Indeed, numbers of cases of chronic constipation are never cured, no matter what therapeutic means are being employed. Preventive measures are, therefore, to be recommended early and carried out with precision.

It is of primary importance to train the child to have a movement regularly every day. Proper habits are often easily formed if the child is put upon the chamber or chair invariably at the same hour. The first few days it may equire local stimulation to defecation (e. g., introduction into the rectum of a small oiled syringe tip). Similar means should be employed also with older children; particularly, with school children who are very apt to suppress Nature's impulse to empty the bowels.

Two main factors are instrumental in the expulsion of the rectal contents: contraction of the abdominal muscles and the diaphragm, and separation or relaxation of the gluteal group of muscles. If the seat of the commode is too high and the aperture in the seat too wide, no support is given to the tubera ischii, the gluteal muscles are crowded together instead of separated, and the descent of the floor of the perineum is much hindered. This impediment to defecation may be obviated by

substituting a low seat on a nursery chair or toilet, or small vessel
for the high one previously used. The child is thus enabled to accom-
plish this act in a squatting posture, which is most favorable to thor-
ough emptying of the rectum.

Correction of diet is, of course, very valuable for the prevention of
habitual constipation, but does not always remedy the trouble. This is
particularly true of cases of very long standing, since here we are
dealing with secondary atony following prolonged distention and enfee-
blement of the intestines. The diet should vary, of course, with the
age of the patient and consistency of the stools. As a rule, the latter are
either small, hard and marble-like, or very large, acholic, and sausage
shaped. In the first case, the dietary should be improved by the addi-
tion of fat and cereal gruels, such as groats and oatmeal. In the second
case, good results are often obtained by reducing the cereals and fats
and by substituting malt cereals. In older children a moderate supply
of cream, malt extract, honey, rye bread, bran, raw or cooked fruit, and
vegetables may remove the difficulty. A glass of cold water on an empty
stomach, and at night before retiring, is often very useful.

Faithful compliance with the suggestions just made very often yields
favorable results. In a certain percentage of cases, however, more active
measures have to be resorted to and it then devolves upon the physician
to select such therapeutic means as will not effect the general wellbe-
ing of the patient. In older children, this indication can most appro-
priately be met by the simultaneous employment of a combination of the
so-called physicochemic procedures, consisting of massage, oil enemas and
hydrotherapy, and occasionally, also electricity. This treatment is more
advantageously carried out in the evening, before the patient goes to
sleep. The child is placed on a hard couch or mattress with head and
thorax raised and legs sharply flexed at the knee-joints and somewhat
rotated outward. The attendant stands on the left side of the patient.
The manipulations are begun at the fossa iliaca sinistra, where the sigmoid
flexure is situated and is frequently found to be a halting place for
hardened feces. With the tips of the fingers of one hand (in older chil-
dren both hands may be used, one hand being placed upon the other),
the attendant makes gentle circular movements along this portion of the
colon and at the same time exerts upon it considerable pressure downward
toward the rectum. Without changing these movements the attendant
slowly ascends as far as the splenic flexure. From here he gradually
returns to the sigmoid. He now begins a new tour, going as far as the
hepatic flexure, and after gradually returning to the starting point he
makes his final trip, reaching the cecum and, in the manner just outlined,
returns again to the fossa iliaca sinistra. These manipulations should

be followed by rhythmical vibratory strokes over the entire abdomen, interrupted by a few pressure movements against the spinal column in the epigastric region. The treatment should last from six to twelve minutes.

Instead of trying the massage, oil enemas, and hydrotherapy separately, it is certainly preferable to employ these three procedures—the *anticostive triad*—simultaneously, since they do not interfere with one another, but, on the contrary, supplement one another in their beneficial effect. Thus, after completing the massage, the little patient is turned upon his left side, and by means of a piston syringe, $\frac{1}{2}$ ounce or more of warm oil is gently injected into the rectum and allowed to remain there. This is followed by the application around the abdomen of a Priessnitz compress, which should be left in place until the next morning. It will almost invariably be found that the patient's bowels will act either during or soon after the treatment, or at any rate, not later than the following morning. A three or four weeks' course of treatment will usually suffice to establish regularity of the bowels, provided the preventive measures suggested before are strictly adhered to. In some very protracted cases of constipation these procedures may be supplemented by the application of the galvanic or faradic current. One electrode is passed successively over different portions of the abdominal wall, and the other electrode is placed upon any other part of the body.

Proctologists frequently advocate divulsion of the sphincter ani as a sure cure of habitual constipation. I am not inclined to be quite as enthusiastic over it, except in cases of constipation due to rectal disease, as, for example, fissura ani, rectospasmus, etc.

Finally, there is a class of cases of chronic constipation which resists all forms of treatment as regards a permanent cure, but may be considerably improved by alternately resorting to the therapeutic measures already enumerated as well as to drugs. In the selection of an evacuant the physician must be guided by the etiologic factors and the individual peculiarities of the case in question. The indiscriminate use of anti-spasmodics (belladonna) as well as the ever-ready ''soothing'' laxatives, is to be strongly deprecated. Of all the laxatives in use, mineral oil is the safest and most efficient. In a child five or six years old, we begin with a tablespoonful once a day and, as regular evacuation is established, we gradually reduce the dose. Temporarily effective and comparatively harmless are also the following remedies: Soap and glycerine suppositories, medicated cocoa butter suppositories (with aloin and belladonna in spastic, or aloin and nux vomica in atonic, constipation), enemas with small quantities of glycerine or larger quantities of soap water; internally, magnesia usta, magnesia and rhubarb, compound licorice powder,

castor oil, extract of cascara sagrada, calomel followed by a mild saline aperient, and, in older children, the standard mineral salts or waters.

Whatever the method of treatment employed, the establishment of a *habit* to move the bowels regularly at a certain time of the day should at all times be our chief aim.

Prolapsus Ani, Prolapsus Recti

If the prolapse is limited to the mucous membrane of the anus, the condition is spoken of as prolapsus ani; if the lower portion of the

Fig. 60.—Prolapsus recti.

rectum protrudes through the anal orifice, it is known as prolapsus recti. In prolapsus recti the protruding part comes down during defecation in the form of a round, or sausage-shaped, glistening, red or bluish red, frequently bleeding mass. In the beginning, the mucous membrane slips back in its place spontaneously, or is easily replaceable and remains there until the next movement; in severe cases, owing

to marked inflammatory thickening, reposition of the mass may be difficult, and if replaced, may immediately prolapse again.

These conditions are very common in young children, the softness of the connective tissue and incomplete development of the muscular system serving as predisposing causes. The ordinary exciting causes are habitual constipation, protracted diarrhea, proctitis, rectal polypus, oxyuris, phimosis, vesical calculus, i. e., conditions in which the act of defecation or urination is attended by pressing, tenesmus, or strangury. Protracted, paroxysmal coughing (e. g., pertussis), by its downward pressure upon the abdominal contents, also serves as an etiologic factor, and prolapsus recti is not infrequently associated with rachitis, probably due to the accompanying muscular debility and constipation.

The diagnosis can readily be made by inspection and digital examination. It is most apt to be confounded with hemorrhoids and rectal polypus. Rectal polypus is the most frequent cause of rectal bleeding in children, and appears at the anus as a dark-red, bean- to cherry-sized, roundish tumor with a bleeding surface. Digital examination usually reveals that the polyp is attached to the rectum, a few centimeters above the sphincter, by means of a short or long pedicle.

Slight prolapse is readily amenable to reposition of the prolapsed mass (oiling and gentle pressure upward with the patient in the knee-chest position) and strapping of the buttocks (in older children only before the act of defecation), in addition to prompt attention to the aforementioned etiologic factors. Severer cases call also for reduction of the local inflammation by occasional painting of the affected area with balsam of Peru or a 2 to 5 per cent solution of nitrate of silver. If these measures fail, the prolapsed mass may have to be treated by punctate or linear cauterization. However, the possibility of subsequent anal stricture, should be kept in mind.

General tonic treatment not rarely succeeds when local procedures fail.

Intussusception

(INTESTINAL INVAGINATION)

Intussusception, or sliding of one portion of the intestines into the other, is an affection principally of infancy and early childhood. The commonest seat of the trouble is the ileocecal region. Thus, the proximal portion of the ileum with or without the cecum becomes invaginated into the colon. Less frequently the ileum slides into the ileum, or a part of the colon into the colon. Occasionally the invagination is multiple and is responsible for the so-called recurrent intussusception (A. Sturmdorf). The immediate results of the invagination are agglutina-

tion of the opposed serous layers and strangulation of the impacted portion of the intestine. If the latter is not soon relieved, gangrene, sloughing and, in a few days, spontaneous discharge of the cast-off piece of intestine occurs—the continuity of the intestine being preserved by end-to-end adhesion.

The disease sets in very suddenly. In the midst of apparently perfect health, or preceded by diarrhea and colic, the child suddenly shrieks from intense pain and presents other symptoms of severe colic which fail to yield to ordinary anticolic therapeutic measures. The pain and restlessness increase, the abdomen, which at first may be normal or even retracted, soon becomes greatly distended, and, accompanied by marked tenesmus, the child passes from the bowels at first small quantities of feces mixed with mucus and blood, and later pure blood, often of a cadaveric odor.

Digital examination discloses blood in the rectum—often long before any is passed with the stools—and if the intussusception is colonic in form, frequently a round mass is observed high up in the rectum. Exceptionally and late the tumor protrudes from the anus. In ileocecal intussusception, inspection and palpation reveal a round "lump" or sausage-shaped mass in the right iliac region, and occasionally a depression below the tumor—owing to displacement of the cecum. The tumor is less pronounced in intussusception of other portions of the intestines, and in some cases can only be detected under anesthesia.

The severity of the onset is no criterion as to the further course of the disease. In a small number of cases the colic suddenly ceases, the child resumes its normal appearance, and exhausted from the agonizing pain, falls into a profound sleep, waking up apparently well—spontaneous improvement or recovery by spontaneous reduction of the invagination has apparently occurred. In such a cure the trouble is not always at an end, for the intussusception is very apt to return after a shorter or longer interval. In another group of cases, after the grave onset, the disease may pursue a milder course. The vomiting, meteorism, and tenesmus abate in their violence; the dejecta lose their bloody consistency, and the colicky pain returns only after long pauses. After three to six days, a piece of gangrenous intestine, the intussuscepted portion, may be discharged per rectum. This process is always fraught with danger, the greater number of these patients dying from general sepsis. The few patients who survive frequently succumb to consecutive chronic gastrointestinal catarrh, with or without intestinal stricture. In the majority of instances, the symptoms grow worse within twenty-four hours from the start of the attack. The vomiting becomes violent and stercoraceous, the pulse feeble, the

extremities cold, the expression of the face pinched, the eyes sunken, and, unless the condition is promptly relieved, the child succumbs within from four to eight days to increasing collapse, not rarely preceded by intestinal perforation and peritonitis.

Fig. 61.—Stick pin in transverse colon giving rise to symptoms of intussusception requiring operation.

At all events the prognosis is very grave. The mortality ranges between from 50 per cent and 80 per cent in cases left alone or treated palliatively. On the other hand, with prompt surgical treatment, the chances for recovery are by far better—about 65 per cent. The best

results (75 per cent) are obtained in cases operated upon within twenty-four hours of the onset of the attack.

The treatment of choice, therefore, is obvious. Early operative interference,—before extensive adhesions and gangrene of the bowels have taken place. Temporizing is fatal. However, before an operation is resorted to, we must be quite certain that we are not dealing with acute peritonitis, appendicitis or intestinal obstruction from other causes—with which diseases intussusception is most apt to be confounded.

DIFFERENTIAL DIAGNOSIS

CHARACTERISTIC SYMPTOMS	INTUSSUSCEPTION	ACUTE APPENDICITIS	ACUTE PERITONITIS	STRANGULATION
Onset	Sudden	Variable	Variable	Sudden
Tumefaction, its seat and nature	Most frequently ileocecal region, occasionally round tumor in rectum	McBurney's point. Rigidity of abdominal wall	Distributed throughout abdomen, also local exudation	Local distention of bowel. Chiefly at abdominal rings
Tympanites ...	Moderate	Absent, at first	Pronounced	Slight
Abdominal pain	Intense, general	Moderate, local	Marked, general	Severe, general
Constipation ..	Late, preceded by frequent muco-hemorrhagic stools	Early	Late	Early
Fever	Slight	High	High	Slight
Collapse	Early	Late	Early	Early

When the services of a competent surgeon are not obtainable, an attempt may be made to reduce the invagination by copious injections of warm (100° F.) water into the bowels, or by air inflation.

For the water injections an ordinary fountain syringe with a rectal tube, suspended about 4 feet above the level of the patient's pelvis, answers the purpose. Two to 4 quarts of water should be used. During this procedure the patient should be kept on his back with his buttocks raised about 1 foot above the level of the shoulders. Occasional inversion of the child, or the Trendelenburg position under anesthesia is useful.

For the relief of pain and arrest of undue peristalsis, morphine and atropine hypodermically; to check vomiting, lavage; to combat col-

lapse, stimulants and external heat. Liquid food that is easily digestible should be given to sustain nutrition. Complications arising, should be treated according to indications.

In view of the obscure causes of this affection, very little can be accomplished in the way of prophylaxis. Avoidance of habitual constipation, of drastic purgatives, and of violent exercise (rapid up-and-down motion) may prove efficient prophylactic measures. Occasionally, intussusception follows typhoid fever, Meckel's diverticulum and severe adhesions secondary to appendectomy. The relationship between invagination and polypoid intestinal growths still lacks authoritative confirmation.

Case Report.—As is usual in acute intussusception, the five-months-old infant under my observation was suddenly seized with pain and vomiting, became very restless and refused to take the breast on which she had been nursed from birth on. As the mother of the baby had at the time been greatly worried over the fate of her husband, who was undergoing an operation for strangulated hernia, she attributed the unexpected illness of her child to some ''nervous'' disturbance of her breast milk. Moreover, on a few occasions the baby had also received a bottle or two of diluted cows' milk, which she thought might have upset her stomach. In addition to this the baby three days before rolled out of its go-cart, head downwards, although apparently without any noticeable bad after-effects. A physician was sent for the same day, and finding the baby suffering from colic, diarrhea and vomiting, ordered a teaspoonful of castor oil, and a rectal irrigation, to be followed a few hours' later by small doses of salol and bismuth. The next day the stools assumed a bloody consistency, and presuming that dysentery was dealt with, he added a few doses of Dover's powder. The opium seemed to relieve the colic, but the bloody stools continued. Alarmed over this condition the family physician kindly invited me to see the case with him. This was about three days after the onset of the vomiting. The patient was drowsy, and its facial features were greatly depressed. Her temperature was 100° F., the pulse slow and feeble, and she seemed entirely free from pain. Her abdomen was slightly distended but on palpation I readily detected an oval-shaped doughy mass in the left iliac region which was very sensitive to pressure. Furthermore, on introducing the finger into the rectum, about two ounces of bloody fluid was forcibly expelled from the rectum along the sides of the examining finger. There could be no doubt as to the diagnosis. The sudden onset, the persistent vomiting (which by the way was not feculent!), the bloody discharge free from feces, the intense colic and above all, the painful mass in the left iliac region, were pathognomonic of intussusception. Dysentery was a plausible diagnosis the first day, but surely not thereafter, when free blood made its appearance. In some cases intussusception may be mistaken for incipient appendicitis, impaction, peritonitis or strangulation, but in none of these cases would we find serosanguinolent and later purely bloody stools. Besides, these diseases have pathognomonic symptoms of their own, which must always be considered in the differential diagnosis. I suggested an immediate operation, and Dr. Lilienthal performed the same within an hour. The laparotomy revealed a colonic invagination at the sigmoid flexure, embracing the entire colon including the cecum. The baby succumbed a few hours later.

Appendicitis, Typhlitis, Perityphlitis

Until recently the prevalence of appendicitis in early childhood was not taken very seriously by the profession at large, and hence, either because of its skepticism, or for want of understanding of the pathology of the disease, a great many cases of acute or chronic appendicitis were either overlooked, erroneously diagnosed or ascribed to "food fever," "cyclic vomiting," and the like. Nowadays, the occurrence of appendicitis in children and even in sucklings is no longer doubted. On the contrary, in view of the frequency with which the vermiform process is found implicated in the course of severe infantile gastrointestinal disease, and its tendency by its relatively greater length and width to favor lodgment of foreign bodies (such as fecal concretions, worms, etc., which act as sources of infection), there is ample reason for the belief that as a whole appendicitis is as common in children as in adults. As in the latter the severity of the disease in infants varies from simple inflammation to fatal gangrene, depending of course upon the type and virulence of the causative bacteria and the promptness with which it is discovered and treated.

Pathologically the simplest form of appendicitis consists of a catarrhal inflammation of the appendix. Its mucosa, and follicles are reddened and swollen, and their secretion is more abundant than normal. The lymphatics of the walls and of the surrounding structures are congested. Gradually the submucous and serous layers become involved and the appendicular lumen narrowed. In mild cases the obstruction in the appendix subsides, allowing the escape of the mucous and bacterial contents, and, with the exception of slight thickening and adhesions, rapid *restitutio ad integrum* takes place.

In more severe cases the obstruction continues, the appendix becomes more and more distended, the mucous secretion purulent, the muscular coat, owing to its effort to expel the appendicular contents, thicker, hypertrophied, while the mucous membrane, as a result of pressure from within the appendix, undergoes gradual atrophy and ulceration. Even in this stage of the disease spontaneous recovery by encapsulation and absorption of the abscess is still possible.

In the majority of instances, however, instead of being absorbed, the purulent content of the appendix gradually, or rapidly, increases in quantity, and finally perforates the overdistended, more or less ulcerated appendix. The escaping pus finds its way where there is least resistance —into the cecum, small intestine, rectum, urinary bladder, gall bladder, diaphragm or into the free peritoneal cavity. The pus may, on rare occasions, also penetrate into the retroperitoneal cavity, or externally, usually in the right iliac region.

Sometimes the inflammation is almost from the start so intense that perforation and gangrene of the appendix, and escape of its virulent contents into the peritoneal cavity occur before a diagnosis can at all be arrived at. In these cases it is not rare to find also old inflammatory adhesions, indicating that the patient had once before gone through an attack of appendicitis (recurrent appendicitis), which probably was mild and had escaped attention.

The great variability in the course and termination of the aforementioned pathologic process can readily be explained primarily by the difference in the virulence of the causal bacteria, no single type of which having thus far proved to be the specific etiologic factor of appendicitis as a whole or of any of its forms. The bacteria found in the inflammatory products of the disease are principally streptococci, staphylococci, *B.* coli communis, the pneumococcus, *B.* influenzæ, etc. It is not at all uncommon for appendicitis to develop in connection with pneumonia, influenza, gastroenterocolitis, etc., thus tending to prove its infectious character. Prominent etiologic factors also are retention of fecal concretions, foreign bodies (pins, fish bones, cherry stones, orange pits), intestinal worms, traumatism, exposure to cold and wet, etc. In a baby eighteen months old, who was operated upon for inguinal hernia, we found seven pinworms and two caraway seeds in a perfectly normal appendix. Male children (possibly because more often exposed to traumatism) are more frequently attacked by appendicitis than female children. Constipation and dyspepsia serve as predisposing causes.

Acute appendicitis may set in very suddenly or be preceded by premonitory signs, consisting of frequently recurring attacks of dyspepsia, with colic and constipation. It is quite probable, however, that the dyspeptic symptoms are in reality the manifestations of recurrent catarrhal appendicitis of very mild type. The appendicitis once established, the little patient stops eating, is nauseated, vomits, and cries because of pain in the abdomen. The latter is more or less rigid. The anorexia is usually complete, and, if the child is forced to eat, the food is sooner or later ejected. Infants may continue taking the bottle or breast, to quench thirst. In very mild cases, nausea may replace the vomiting, but the latter symptom is always present in moderately severe cases and is quite severe in grave appendicular involvement, especially when the peritoneum is implicated. Pain, spontaneous and on pressure, is invariably present during an attack, but it varies greatly in severity irrespective of the pathologic condition of the appendix. Sudden cessation of pain often signifies mortification of the underlying structures, and, hence, is to be looked upon as a bad omen. Young children are usu-

ally unable to localize the seat of the pain they are suffering from; little reliance, therefore, should be placed upon its localization. On the other hand, pressure pain can readily be elicited, which, as a rule, is most intense over the region of the appendix, and which in children does not always correspond with "McBurney's point"—the appendix is often situated either higher up or lower down in the pelvis. Sometimes, even infants indicate the presence of pressure pain by attempting unconsciously to ward off the examining hand, by placing their little hands over the most painful spot. Rigidity of the abdominal wall forms a pathognomonic sign of the disease, and proves of great help in the diagnosis of appendicitis to one familiar with the peculiar sense of resistance of the abdominal wall to pressure. As a rule, the abdomen is distended, but it may also be contracted and as hard as a board. On gentle palpation the rigidity yields sufficiently to permit the detection of tumefaction—the underlying thickened appendix in catarrhal appendicitis, or the variously sized, hard or doughy, immovable mass in appendicular abscess. In rare cases the tumefaction may be seen to project beyond the normal level of the skin, or be felt in the rectum; a digital examination, therefore, should never be omitted. As a rule, the patient suffers pain when his right leg is extended forcibly, and in walking he usually "favors" this leg and often puts the right hand upon the abdomen to prevent shaking of the underlying structures. Appendicitis is ordinarily associated with complete constipation; the attack may, however, be ushered in by diarrhea, or, rather, pseudodiarrhea, since the stool is derived chiefly from the lower part of the colon, superinduced by the sudden irritation within and about the appendix. As the disease advances, in consequence of pressure by the growing tumefaction in the pelvis, there may be severe tenesmus (as well as strangury) with or without a bloody discharge,—a symptom which is very apt to mask the diagnosis. The temperature is moderate, from 101° F. to 103° F. in catarrhal appendicitis, and as high as 105° F. in abscess formation. In favorable cases the pulse and respiration agree with the rise or fall of the fever. Low temperature with a high, feeble pulse and complete cessation of pain are considered a bad omen, an indication of profound sepsis or perforation of abscess.

Diagnosis.—Cases presenting the aforementioned typical symptoms of appendicitis can be diagnosed as readily in the child as in the adult. In fact, owing to the thinness of the infantile abdominal wall, and the proportionately large size of the appendix, it is usually not difficult to palpate an inflamed appendix unless it be—as it sometimes happens—misplaced somewhere beyond the reach of palpation. On the other hand, there is often considerable difficulty to differentiate an

appendicitis pursuing a very violent course with marked tympanites, shock and collapse, from a grave attack of acute gastroenterocolitis, pneumonia, typhoid with perforation, intussusception, perinephritic abscess, hernial strangulation, severe purpura hemorrhagica and the like. Even in such cases careful analysis of the typical symptoms of the respective diseases rarely fails to lead to a correct diagnosis. Chronic appendicitis with recurrent acute exacerbations can usually be differentiated from renal calculi by x-ray examination and cystoscopy.

Course and Termination.—The severity or mildness of the onset of an attack of appendicitis bears no positive relation to the further course of the disease. After the inflammatory process has, so to say, localized itself, which occurs usually within the first twenty-four or forty-eight hours, the physician is able in the majority of instances to conclude what sort of a case he is dealing with. By that time he will find that in catarrhal appendicitis the vomiting has partially or entirely ceased, the pain diminished, the abdominal rigidity lessened, and the tumefaction become less palpable. The child is able more easily to move about in bed, to have a few hours of comfortable sleep, occasionally to expel flatus, and to express a desire for food. Uneventful recovery may now take place within ten days, *i. e.*, as far as subjective signs are concerned. In the majority of cases some morbid anatomic changes remain in the appendix and adjacent structures, *e. g.*, inflammatory adhesions, kinking, constriction of the lumen, etc. The region of the appendix thus remains a *locus minoris resistentiæ* for life, subject to recurrent attacks of inflammation and its sequelæ.

Sometimes after an apparently benign course of a few days' duration, either without discernible cause or as a result of gross errors in diet, undue exercise, and the like, there is a sudden change for the worse. The symptoms, spoken of as occurring with the onset, return, sometimes even in more pronounced form; the patient vomits, has chills, headache, severe pulling and throbbing pain in the abdomen. The temperature rises, the pulse increases in frequency and tension, respiration is quick but superficial (the patient is afraid to cough or take a deep breath owing to the increase of the pain with the descent of the diaphragm); the child is restless and sleepless, lies principally on his back with his right leg flexed (attempt to extend it aggravates the pain), and cries with pain on being moved about. Palpation reveals a distinct oblong tumor, the distended appendix, which is very tender, and gives rise to a gurgling sound on pressure. This physical sign is often absent in the so-called retrocecal appendical abscesses! If the disease is not checked by operation, the indurated mass enlarges, loses its circumscribed character, becomes more doughy in consistency, and

dull on percussion; in short, it presents unmistakable signs of a fluid content—an abscess. This clinical picture of suppurative appendicitis does not by any means follow only the catarrhal variety; on the contrary, quite often it is in full development within the first two or three days of the disease, and if the abscess is not promptly opened, it bursts, often giving rise to general peritonitis and quick death. More rarely the accumulation of pus occurs very slowly and gradually, and even remains in abeyance for a period of weeks or months, during which time the abscess becomes walled off from the general peritoneal cavity by inflammatory adhesions, and may finally be absorbed, or, with recurrent attacks of appendicitis, perforate the sac and wander into any of the neighboring structures, sooner or later leading to the grave symptoms previously spoken of.

In another group of cases—fulminating, gangrenous appendicitis—the symptoms are extremely alarming immediately from the beginning of the attack. In the midst of apparent good health, or preceded by slight malaise, vomiting, colic, prostration and collapse, following one another in rapid succession, and often without palpable local appendicular tumefaction, or other signs pathognomonic of appendicitis, the typical picture of general septic peritonitis is in its full sway, —sometimes within twenty-four hours (usually after from three to five days) carrying the little victim to the grave. In such cases postmortem examination reveals either preexisting infection of the peritoneum, or sloughing of a gangrenous appendix, involvement of adjoining blood vessels (thrombophlebitis) and general sepsis (pyemia).

Treatment.—In view of the uncertainty of the course of the disease, every case of appendicitis should sooner or later be operated upon. This opinion is in accord with that held by the best modern clinicians. The profession is still divided, however, on the question of the time when operative procedures prove most propitious for the patient's uneventful recovery. In solving so difficult a problem, the physician must be guided (1) by the condition of the patient, and (2) the progress of the disease.

1. *The Condition of the Patient.*—It certainly would be folly to operate on a child in a moribund condition, or on one synchronously suffering from a systemic fatal disease *per se, e. g.,* miliary tuberculosis, diabetes, grave heart or kidney disease, and the like. An operation should, if feasible, be deferred in infants under six months of age, because of the lack of resistance of the patient, and in view of the fact that in very young infants spontaneous recovery (at least temporary), by absorption of the pus, or rupture of the abscess in the rectum, is by no means rare.

2. *Progress of the Attack.*—Mild catarrhal appendicitis, with the first attack, progressing favorably during the first four days, may be left alone until the quiescent stage, when the appendix should be removed. Severe or recurrent catarrhal appendicitis, failing to improve after the fourth or fifth day or showing incipient symptoms of suppuration (increased leucocytosis), should be operated upon at once; or, if for some reason an operation cannot be undertaken, it should be treated medically for a week or ten days longer, until the abscess has become circumscribed and encapsulated, when an operation should be performed without further delay. The same rule applies also to all cases of slowly developing suppurative appendicitis, the physician being constantly on the guard, however, for sudden threatening symptoms of perforation,—in the latter event demanding prompt surgical interference. Finally, an immediate operation is imperative in all cases of perforative and gangrenous appendicitis, procrastination proving almost invariably fatal.

When a patient is seen early, it is advisable to administer one dose of castor oil or calomel with bicarbonate of soda, to wash out the stomach (in the presence of vomiting) and intestines—to clean the alimentary canal of its contents. This should be followed by an occasional administration, in the form of suppositories, of very small doses of codeine or opium, to arrest peristalsis and to keep the child perfectly at rest and free from severe pain. No medication by mouth. During the acute stage of the disease, the constant application of ice is useful to relieve pain and arrest rapid progress of the inflammation. Thirst should be relieved by *small* quantities of water or tea; and so long as anorexia exists, no attempt at forced feeding should be tolerated. An occasional teaspoonful of milk or broth will prove sufficient to sustain life for days. Any indiscretion in the diet is hazardous. I have frequently observed recurrence of an attack after partaking of cold drinks or ice cream. More liberal feeding may be practiced after subsidence of the acute symptoms, after repeated escape of flatus or of partly formed stool. Even then extreme caution is commended, limiting the dietary to slowly increasing quantities of milk, broths; albumin water; in older children, fresh soft-boiled eggs, milk toast, small portions of fine cereals, etc. For marked tympanites, atropine and morphine hypodermically. Stimulation by means of strychnine and normal saline solution, both subcutaneously, should be resorted to in accordance with indications. As the patient recovers, medication in the form of stomachics, intestinal antiseptics and laxatives may be administered by mouth, and the supply of nutritious food increased, so as to heighten the patient's vitality for an early operation. Children convalescing from an

attack of nonoperated appendicitis should not be taken to any resort
where a competent surgeon is not within immediate reach. Danger al-
ways lurks behind a diseased appendix.

Peritonitis Acuta

Acute, nontuberculous peritonitis is of rare occurrence in children.
The primary form is usually due to infection of the peritoneum by the
B. coli communis, streptococci, staphylococci, or pneumococci, or by a
combination of them. It is occasionally also encountered as a result of
direct violence or secondarily in connection with infectious diseases
e. g., typhoid fever, scarlatina, diphtheria, pneumonia, dysentery, vulvo-
vaginitis, appendicitis and extension of other pus foci. In the new-
born it not rarely forms a partial manifestation of sepsis (*q. v.*).

Acute peritonitis usually sets in with very acute symptoms: excessive
pain and tenderness of the abdomen, rapidly developing tympanites, at
first often diarrhea, later constipation, scanty urination, or complete
anuria; sometimes distinctly localized exudation; which may be dis-
cerned by dulness in the flanks; high fever, especially during the first
few days, more particularly in the perforative forms, and a feeble, rapid,
and very poor pulse; dry and brown tongue, anxious and pinched ex-
pression of the face, and, as the disease progresses, collapse. As a rule,
marked leucocytosis prevails. The course of the disease varies. Hyper-
acute peritonitis ends fatally usually in two or three days; moderately
severe cases may last a week, and then terminate either in death or in
gradual recovery. To the latter class belong also the cases usually of
pneumococcus origin in which the pus becomes encysted, and breaks
through the umbilicus, rectum or bladder.

In a case (girl four years old, ill five weeks) I recently saw in con-
sultation, the onset was sudden with vomiting, pain, and high fever.
These symptoms subsided after a week, leaving behind very marked ab-
dominal distention, slight, irregular fever, constipation and distinct
flatness over the entire lower abdomen. Palpation also revealed very
pronounced enlargement of the spleen. I concluded that we were deal-
ing most probably with a secondary purulent peritonitis and suggested
laparotomy, which would prove beneficial also were the case to turn out
to be tuberculous peritonitis. This was done the following day. Over a
quart of freely-flowing, foul-smelling pus escaped through the abdominal
opening and the patient made an uneventful recovery. Apparently the
peritonitis was of appendical origin.

At all events the prognosis is very grave. It is almost always fatal
to the newborn, and in cases resulting from intestinal perforation.
Traumatic peritonitis offers the most favorable outcome, and local peri-

tonitis with encapsulated abscess often yields to prompt and suitable treatment. Protracted cases may be complicated by pleurisy, pericarditis, meningitis and general pyemia.

The *treatment*, of course, depends entirely upon the underlying condition. It is justifiable to recommend an operation (laparotomy) in all cases of acute general peritonitis that fail to respond to medical treatment within forty-eight hours, and in those resulting from perforation of an abdominal viscus, *e. g.*, appendix, intestinal perforation in typhoid. (For "differential diagnosis," see p. 267.)

The medical treatment consists of perfect rest for the body and immobilization of the intestine. This may be secured by the hypodermic administration of morphine (1/60 grain for a child two years old) and atropine (1/1000 grain), the application of an ice bag or light turpentine stupes to the abdomen, and discontinuance of any nourishment until vomiting has completely ceased. Vomiting is best arrested by lavage, sodium bicarbonate, bismuth subcarbonate, or minute doses (m. 1/30) of tincture of iodine. After arrest of vomiting, feeding may very cautiously be resumed. Breast-fed babies may again be put to the breast and bottle-fed babies should receive small quantities of milk, gruel, beef juice, Tokay wine, champaign, and, if improvement continues, a light mixed diet. For excessive tympanites, the long rectal tube may be tried, allowing it to remain *in situ* for hours at a time. Or the saline "Murphy drip" the latter having the effect also in draining the abdomen of its toxic products and acting as a stimulant. Cases running a protracted course sometimes do well on daily local inunction of ung. hydrargyri (½ dram), and the iodides internally. Localized abscesses should be incised and drained. In slow convalescence, a sojourn at the seashore will prove beneficial. (For "Tuberculous Peritonitis.")

Intestinal Worms

Worms gain entrance into the human system chiefly through the ova, either consumed with food and water, or carried to the mouth by means of the fingers. We distinguish the following varieties of worms:

(a) *Oxyuris Vermicularis (Seat-, Thread, or Pinworm)*.—Small, white, thread-like, freely movable worm, ¼ to ½ inch in length. Its chief seat is the rectum where it causes intense itching. It may also infest the colon, cecum, appendix and vagina (vulvovaginitis).

(b) *Ascaris Lumbricoides (Roundworm, Nematoda)*.—Cylindrical, reddish gray in color, from 4 to 10 inches in length. It resembles the earthworm in form. Its chief seat is the small intestine, but it may mi-

Fig. 62.—Oxyuris vermicularis. Female and male. (After Leuckart.)
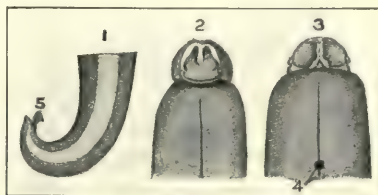
Fig. 63.—Ascaris lumbricoides. (1, Tail of male; 2, and 3, mouth—anterior and posterior; 4, excretory pore.)
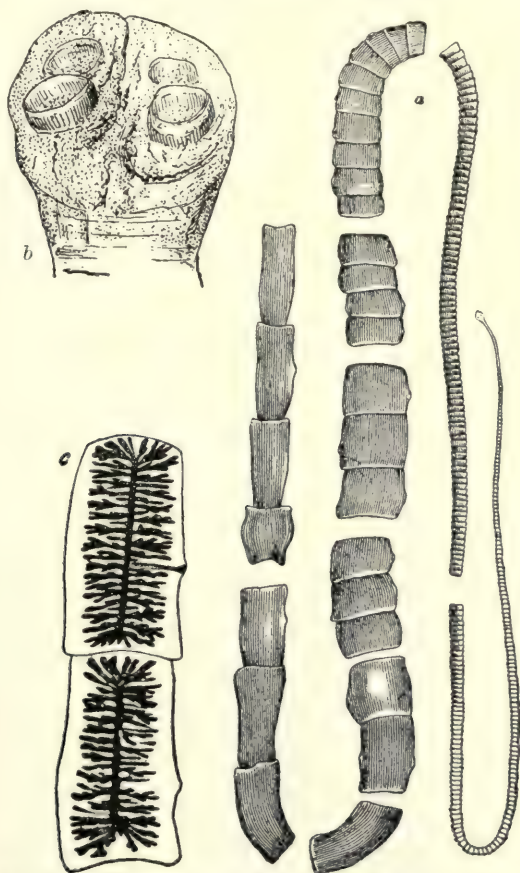
Fig. 64.—Tenia saginata. a. Natural size of the worm at different sections. b. Head (with pigment canaliculi). c. Proglottides. (Partly after Leuckart and Lenhartz, F. A. Davis Co.)

grate to the stomach, gall bladder (icterus), throat, etc., in the latter location occasionally producing attacks of suffocation.

(c) *Teniæ (Tapeworms, Cestoda)*.—They are segmented worms of variable size. They inhabit the intestine and develop by budding.
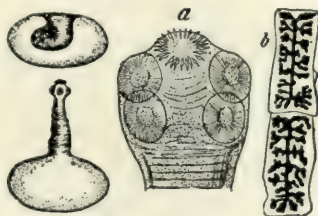
Fig. 65.—Tenia solium. *a*. Head. *b*. Proglottides. (After Leuckart.)
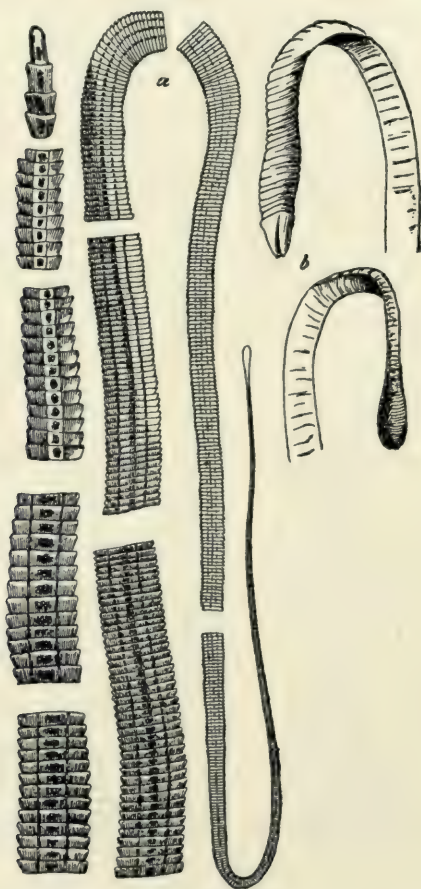
Fig. 66.—Bothriocephalus latus. *a*. Worm, in sections; natural size. *b*. Head; lateral and front views. (After Leuckart.)

(*d*) *Tenia Mediocanellata s. Saginata (Beef Tapeworm).*—It is several yards long. The head presents at its middle a pit-like excavation and four anterior suckers.

(*e*) *Tenia Solium (Pork Tapeworm).*—It is shorter than the former. It is provided with four suckers, one proboscis, and a wreath of hooklets. After invading the human stomach, the liberated embryos may wander to various portions of the body (skin, heart, brain, and eyes) and there develop into small vesicles (cysticercus) and lead to serious disturbances.

(*f*) *Bothriocephalus Latus (Fish Tapeworm).*—Several yards long, possesses about 3,000 segments, a flattened head with two shallow suction grooves. May be the cause of severe anemia.

(*g*) *Tenia Nana.*—About 1 inch long, possesses a head with four suckers and a wreath of hooklets. May cause stubborn diarrhea.

(*h*) *Tenia Cucumerina s. Elliptica.*—From 5 to 15 inches long; develops from swallowing dog ticks which infest the hair of dogs and cats.

(*i*) *Tenia Echinococcus.*—It inhabits the intestines of the dog. The latter transmits the ova to the human gastrointestinal tract through the mouth, by licking, etc. The embryos develop chiefly in the liver and lungs, forming cysts.

Symptomatology.—In times bygone the laity looked upon intestinal worms as the source of all ills, and even the physician was frequently inclined to hold the same view. As a matter of fact, worms, with but few exceptions, rarely produce very serious disturbances. Indeed, numerous round- and tapeworms may infest the human intestines often without any indication of their presence until accidentally discovered in the stools. Among the signs which are otherwise said to indicate their presence are the following: A pale complexion, black rings under the eyes, *fetor ex ore,* capricious appetite, picking at the nose, recurrent urticaria, colic, headache, vertigo, apathy, mydriasis, pavor nocturnus, grinding of the teeth, and dry cough. Some authors claim to have observed divers neuroses, convulsions, chorea, trismus, epilepsy, amblyopia, strabismus, and the like. The majority of the reported cases of this sort, however, do not bear close scrutiny and are readily traceable to other causes. The actual harm done by some of the worms has been mentioned under each heading.

Diagnosis.—The diagnosis can readily be made by macro- and microscopic examinations of the stools and sputum (echinococcus hooklets) for worms or their ova. The finding of intestinal parasites may be facilitated by the administration of anthelmintics.

Treatment.—Santonin and calomel act very efficiently in thread- and roundworms.

> ℞ Santonini,
> Hydrargyri chloridi mitis....āā gr. vj | 0.4
> M. et div. in pulv. no. vj.
> S.—One powder to be given every morning, on an empty stomach for a child three years old.

To expel teniæ the following is a very useful combination:

> ℞ Ext. aspidii fl.................3iij | 12
> Emulsi chloroformi ....3iv | 15
> M. Emulsi amygdalarum ....q. s. ad ℥ij | 60
> S.—Two teaspoonfuls as a dose for a child three years old, to be administered as follows:

The day before the diet should be restricted to fluids. In the evening the patient is given a few pieces of salt herring, followed an hour later by a purgative (castor oil or calomel). The next morning the male fern should be administered on an empty stomach, followed within half an hour by a dose of castor oil or calomel. If only part of the tapeworm escapes, and the other part remains inside, the torn end should by means of adhesive plaster be fixed to the buttocks, and another dose of the anthelmintic and oil administered until the rest of the worm has been expelled.

The effect of anthelmintics by mouth is greatly enhanced by enemas of soapsuds and turpentine (½ dram to 1 pint) or a decoction of quassia wood (1 ounce to 1 pint). Quassia injections are very useful in pinworms, especially if followed by local application of gray ointment In older children the fluid extract of male fern may preferably be given in capsule form. The rare attacks of asphyxia from round-worms, previously spoken of, are best relieved by turpentine administered by mouth (on lumps of sugar) or by rectum, and prompt expulsion of the worm by santonin.

Ankylostomiasis, Uncinariasis
(Hookworm Disease)

Although prevailing in this country for many years past, this affection has only recently, principally through the efforts of Dr. Charles W. Stiles, received due recognition as the "American murderer." It is practically endemic throughout the South, but is met with sporadically also in other states of the Union.

The disease is caused by the hookworm which infests the human body either through the mouth (by swallowing of infected water or food), or through the skin, especially the skin of the feet (the larvæ

of the worm gradually entering the circulation), and ultimately settles in the upper portions of the small intestines.

The hookworm comprises two species: *Ankylostoma duodenale* (old-world species), which is endemic, especially in Italy and Egypt, and
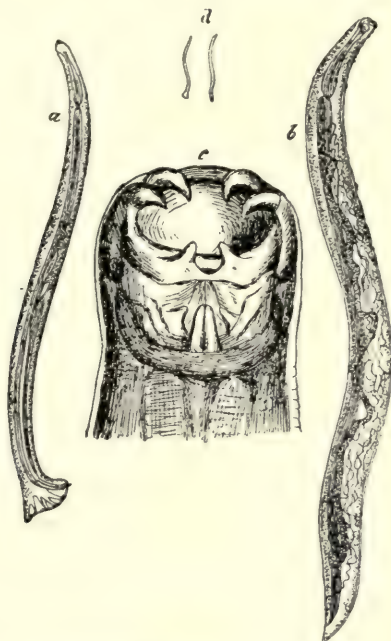
Fig. 67.—Ankylostomum duodenale. *a.* Male. *b.* Female. *c.* Head. *d.* Natural size. (After Leuckart.)
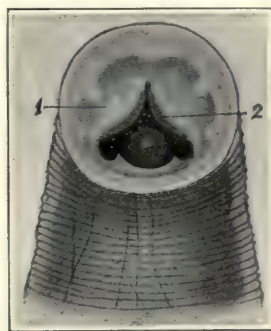
Fig. 68.—Uncinaria Americana. (*1*, mouth capsule; *2*, mouth cavity.)

Uncinaria americana or *Necator americanus* (the new-world species). Both species measure from about ⅓ to ⅔ inch in length (the females somewhat larger than the males), but while Ankylostoma carries on its

head four hook-like teeth on the ventral side and two smaller vertical teeth on the dorsal side, the Uncinaria has a dorsal pair of prominent semilunar plates or lips, and a ventral pain of smaller plates of similar nature.

By means of its armed mouth the worm fixes itself to the intestinal mucosa, producing minute erosions and hemorrhagic spots, and sooner or later a more or less severe catarrhal process in the alimentary tract. It is still a matter of diversity of opinion whether the uncinaria feeds on the epithelial cells of the mucosa or upon blood. However this may be, the blood certainly undergoes marked changes, in severe cases, resembling the blood findings of primary pernicious anemia. Leukocytosis with eosinophilia is the rule. Very soon other organs of the body are affected, especially the liver and spleen.

Postmortem examination usually reveals fatty degeneration of the liver; softening of the spleen and paucity in lymphoid elements; nephritic changes in the kidneys; pallor of the lungs; flabbiness of the heart, and anemia of the brain and effusion into the ventricles.

Hookworm disease is most destructive in the young. Usually dermatitis of the feet and legs forms the first symptom. Children remain stunted in physical and mental development, they look tired, old, apathetic, and owing to the puffiness of the face not rarely resemble cretins. The skin is sallow, the fingernails and the scleræ are white or bluish-white. They suffer from palpitation of the heart, dyspnea, headache, dizziness, tinnitus, nausea, occasionally vomiting and abdominal pain. The appetite is either poor or voracious, often accompanied by a desire for unnatural food (pica), eating of earth, dirt, rags, etc. With increasing anemia there is frequently dropsy in the subcutaneous tissues and serous cavities—the edema often masking the emaciation and flabbiness of the body musculature.

Occasionally the disease runs quite a rapid course, the patient dying from exhaustion within a few weeks.

The diagnosis of hookworm disease is based upon a macroscopic and microscopic examination of the stools for the worm and its ova.

Treatment.—Thymol acts specifically in this affection. It may be administered in an emulsion with acacia or, in older children, in the form of capsules, the thymol crystals being first triturated with sugar of milk. The following mode of administration is recommended: Late in the afternoon the patient receives 2 grains of calomel (no castor oil) and the next morning 1 dram of Epsom salts. After the bowels have thoroughly acted, 5 or 10 grains of the thymol is given on an empty stomach, and, if indicated, the dose is repeated after an hour.

The patient is kept in bed, without food, until late in the afternoon. Some clinicians recommend oil of chenopodium instead of thymol.

The feces should again be examined for uncinaria after the lapse of from two to four weeks.

DISEASES OF THE LIVER

Icterus Catarrhalis

(CATARRHAL JAUNDICE)

Catarrhal icterus (catarrh of the ductus choledochus) occurs as frequently in children over four years of age as in adults. It is comparatively rare in infants, except in the newborn. (See p. 231.) As a rule, it is caused by and associated with gastroduodenal catarrh, and begins with coated tongue, anorexia, nausea, vomiting, and slight rise of temperature. (In another group of cases which is of microbic origin (*epidemic icterus* or Weil's disease), the onset is sudden, with high fever, apathy, delirium, headache, and vomiting, so that before the appearance of the icterus cerebral disease is first thought of.) In a day or two it is usually found that the urine is brownish yellow (bile stained), the feces are gray and clayey, and the conjunctivæ, scleræ and skin yellow in color. This pathognomonic group of symptoms increases in intensity up to about a week, and then begins to diminish, first with clearing of the urine. The pulse is usually retarded, about seventy beats to the minute when the child is at rest. Palpation and percussion reveal tenderness over the stomach and liver, and occasionally some enlargement of the latter. This is particularly the case in catarrhal jaundice running a protracted course.

The prognosis is favorable and under suitable treatment the symptoms ordinarily subside within from ten to fourteen days. The *treatment* consists of restriction of diet to thin soups, albumin water, skimmed milk, tea and toast, boiled fish or chicken, and similar, easily digestible food, free from fat (no cream, eggs or pastries!). Gradual return to a heavier diet. Medicinally, a few small doses of calomel and bicarbonate of soda, and daily intestinal irrigation (with 2 quarts of water at 90° F.) will usually suffice to arrest the disease. Pancreatin, rhubarb and soda mixture, and sodium salicylate are useful remedies, and prolonged warm alkaline baths (1 pound of bicarbonate of soda to the bath) hasten recovery in chronic cases.

Diseases of the Parenchyma of the Liver

Primary disease of the parenchyma of the liver is extremely rare in children under twelve years of age, since its principal cause—alcoholism—is practically unknown in young children. On the other hand,

secondary involvement of the liver is not infrequently met with in connection with syphilis, tuberculosis, chronic suppurative processes, malaria, rachitis, valvular heart disease, protracted gastrointestinal disease, and infectious fevers. In these conditions the symptomatology is the same as in adults.

Cirrhosis of the Liver

1. **Atrophic Cirrhosis.**—After a prodromic stage of several weeks, consisting chiefly of gastrointestinal disturbances, emaciation, tympanites, ascites, slight enlargement of the spleen, and dilatation of the abdominal veins gradually complete the clinical picture of the disease. The atrophy of the liver usually sets in insidiously, as a result of gradual hardening and contraction of the connective tissue. The course of the disease is shorter in children than in adults. Hemorrhages from the stomach and nose and into the skin not rarely occur toward the end of the disease, and progressive ascites hastens fatal termination.

Case Report.—C. H., male, six years of age, was of healthy German parents. When barely a few months old he was frequently given a taste of beer, to initiate him, as it were, in the national custom. He liked it immensely from the start, and as he grew older this beverage served very handily as a prompt pacifier to subdue his ungovernable temper. He was breast fed up to twenty months, and when he was weaned he stubbornly refused to drink cow's milk. Beer again proved the most alluring substitute. It was given to him either cold, mixed with the yolk of an egg, or in the form of "*Bier-Suppe*," *i.e.*, boiled beer with small squares of toasted rye bread. The boy did exceedingly well for several years. At last he began to suffer from frequent attacks of indigestion. Every article of food was blamed for his upset stomach except the beer; and, as on the advice of the family physician, his diet was restricted to the limit barely to sustain his life, beer again stood him in good stead in times of distress. When I saw him he was greatly emaciated. His abdomen was immensely enlarged, very tense and traversed by large tortuous veins, and revealed the presence of a large quantity of fluid. It was utterly impossible to palpate the intraabdominal organs. I withdrew about three pints of clear, yellowish fluid, and was then enabled to determine the absence of any growth or tumefaction in any portion of the abdominal cavity, and the great reduction in the size of the liver.

2. **Hypertrophic Cirrhosis.**—This disease is characterized by considerable enlargement of the liver, pronounced icterus, very marked enlargement of the spleen, and a protracted course. Ascites is absent until very late. The children usually remain stunted in growth. The liver is of very hard consistence.

3. **Congestive Cirrhosis (Cardiac Cirrhosis, Cardiotuberculous Cirrhosis).**—Pathologically it is characterized by hypertrophy of the liver and spleen, obliteration of the pericardium, and by tuberculous pleuritis and peritonitis. Intense ascites forms the principal clinical symptom.

4. **Sugar-Cake or Sugar-coated Liver (Pericarditic Pseudocirrhosis of the Liver—Pick's Disease).**—This form of liver disease is closely allied to the former variety. It is a progressive, incurable affection of unknown etiology.

Treatment.—Since small quantities of spirituous liquors have proved to be the cause of quite a few cases of hypertrophic cirrhosis of the liver in children, it is essential to interdict its use in children, unless intended for temporary therapeutic purposes.

The iodides and mercury should be given a fair trial in all forms of cirrhosis irrespective of cause. The ascites may be relieved by tapping, if diuretics, cathartics and heart stimulants fail to do so. Bland diet. Sojourn at the seashore.

Acute Yellow Atrophy

Its course is very violent, sometimes ending fatally within a few days. The symptomatology is the same as in the adult: high fever, icterus, hematemesis, bloody stools, cerebral symptoms.

Fatty Liver

Anemia and emaciation are the principal symptoms. The liver is often moderately enlarged. The stools are grayish, pasty. The course is chronic.

Amyloid Liver

It is often associated with amyloid degeneration of the spleen and kidneys, and secondary to some wasting disease, especially chronic suppurative processes in the bones and joints. The hepatic and splenic dulness is enlarged, but pain on pressure, jaundice, or ascites are absent, unless the portal circulation is interfered with by enlargement of the glands in the portal fissure.

Attention to the cause, and to the dietetic and hygienic measures, may prove effective to arrest the degenerative process.

Abscess of the Liver

This condition is occasionally observed in children, most frequently as a result of extension of septic processes from neighboring structures, e. g., suppurative appendicitis, phlebitis umbilicalis, typhoid or dysenteric intestinal ulceration. It may follow traumatism, invasion by roundworms, suppuration of echinococcus cysts, or of the mesenteric glands. The abscess may perforate into the thorax, intestines, or externally.

Symptomatology.—Chills, hectic fever, tenderness over the liver; sometimes fluctuation and pus on aspiration.

Treatment.—Free incision and evacuation of the pus as soon as the diagnosis has been established.

Tumors of the Liver

Benign, as well as malignant, tumors of the liver are occasionally observed in young children and even in the newborn. Cystic degeneration is most common, and cases of carcinoma, adenocarcinoma and, more rarely, sarcoma are on record. These growths should not be confounded with gumma of the liver—a positive Wassermann reaction and the effect of specific treatment being most decisive in the diagnosis.

DIFFERENTIAL DIAGNOSIS

	LIVER ABSCESS	HYDATID CYST OF THE LIVER.	PLEURISY WITH EFFUSION	SOLID TUMOR OF THE LIVER
Chills	Marked	Absent	Slight	Absent
Fever	Hectic	Absent	Moderate	Absent
Tenderness	Marked	Absent	Absent	Moderate
Icterus	Slight, early	Late	Absent	Marked, late
Fluctuation	Moderate	Pronounced "hydatid vibration"	Absent, diffuse flat area, uninfluenced by inspiration	Absent
Dulness	Highest in mid-axillary line	Highest in mid-axillary line	Lowest in mid-axillary line	Irregular
Aspiration reveals	Pus	Nonalbuminous fluid with "hooklets"	Albuminous fluid which coagulates on boiling. Pus in pyothorax	Blood
Lung symptoms .	Absent	Absent	Present	Absent

CHAPTER VI

DISEASES OF THE RESPIRATORY SYSTEM

General Remarks

The inherent frailty of the infantile respiratory tract is very conducive toward its morbidity. The nasopharyngeal passages being very narrow and winding—intended to halt air impurities and to moisten and warm the inspired air before its entrance into the larynx —functionate to their own detriment in localities where the air is dust-, smoke- and dirt-laden, and where atmospheric changes are many and marked. Thus, the child being unable to clear its nose, the detained foreign bodies irritate the delicate, highly vascular mucous membrane, before long forming a nidus for bacterial invasion. As we shall see later, "a cold in the head" is quite common in infants, and, while *per se* harmless in its immediate effect, is often serious in its remote results. The local congestion by its repeated recurrence produces a *locus minoris resistentiæ* not only of the mucous membrane of the nose, but, by extension and persistence, of the inflammatory changes (hypertrophy), of the pharynx and adenoid tissue as well. With ensuing nasopharyngeal obstruction breathing now proceeds principally through the mouth; the air no longer undergoes the preparatory process of filtration, moistening and warming, but reaches the larynx in its impure, irritating state, sooner or later giving rise to a catarrhal inflammation of the larynx and neighboring structures. This condition is soon aggravated by the continuous affluxion of foul nasopharyngeal secretion, and by the inability of the little patient to clear its throat by forceful expectoration. Furthermore, the thorax being short and narrow, its musculature thin and feeble, and the heart and thymus gland comparatively large, the more or less compressed lung is greatly hampered in free aëration and in ridding its distantly located portions of the obnoxious inflammatory products. Hence the pertinacity of apparently insignificant pulmonary lesions, the frequency of unresolved pneumonia and pyothorax, and the insidious development of asthma, bronchiectasis and emphysema. As the child grows older, the nasopharyngeal tract larger, the thoracic cavity more spacious and, synchronously, the respiratory function more forceful, there is a corresponding reduction in the frequency and persistency of respiratory disease, notwithstanding, or, perhaps, because of the increased exposure of the child to atmospheric changes and infection.

DISEASES OF THE NOSE AND THROAT AND EAR

Rhinitis Acuta

(Coryza)

Acute coryza is a frequent affection of childhood. It may occur primarily as a result of bacterial infection or follow exposure to thermic, mechanic or chemic irritation, or set in in association with measles, influenza, scarlatina and diphtheria. The infectious variety often occurs in epidemic form.

Primary coryza, if mild in character, gives rise to sneezing, slight rise of temperature, anorexia, etc. On the other hand, if severe in form, especially in infants, it usually begins with vomiting, fever, occlusion of the upper air passages by mucous or mucopurulent secretion, secondary conjunctivitis, and sometimes with convulsions. Owing to thickening of the nasal mucous membrane there is partial or total obstruction to nasal breathing, giving rise to interference with suckling, dyspnea, and even acute attacks of asphyxia. The latter are prone to occur especially in the newborn who are very apt to ''swallow'' the tongue.

Every case of acute rhinitis associated with severe local (pseudomembranous deposit) and systemic (vomiting, rapid loss of strength) symptoms should arouse the suspicion of being diphtheritic or scarlatinal in character.

Acute rhinitis is not rarely complicated by otitis, laryngitis and bronchitis and exceptionally by sinusitis (in older children). The prognosis is generally good, although in young infants convalescence is slow.

Treatment.—Avoidance of exposure to all atmospheric changes, even as regards temperature in the room. Cleansing of the nostrils by repeated instillation of a few drops of a 2 per cent solution of bicarbonate of soda, alternated with lukewarm mentholated olive oil or albolene. Careful feeding, if necessary, by the spoon. As measures of temporary relief, we may recommend local applications of atropine ($\frac{1}{4}$ per cent), cocaine (1 per cent), or suprarenal solutions ($\frac{1}{10}$ per cent), and camphor and the salicylates and quinine internally. There should be more or less strict isolation of the patient. Attention should be paid to constitutional symptoms. Serum therapy, whenever it is indicated (diphtheria).

R̤   Natrii salicyl ....gr. xii | 0.8
　　　Pulv. camphoræ ....gr. iii | 0.2
　　　Chocolate ....q. s. |
　　M. Div. in pulv. no. iv.
　S.—One powder every two hours for a child three years old.

Rhinitis Chronica

(NASAL CATARRH, OZENA)

It is characterized by marked congestion and thickening of the nasal mucous membrane and hypersecretion—*hypertrophic rhinitis,* or by atrophy of the various layers of the mucous membrane and foul-smelling incrustation—*atrophic rhinitis, ozena.* The latter form is rarely observed in children under ten years of age. In the nursling it is often due to lymphatism or more rarely to hereditary syphilis (syphilitic rhinitis).

Chronic rhinitis is usually the result of repeated attacks of acute coryza or other affections of the nasopharynx associated with nasal hypersecretion and obstruction to free nasal breathing (adenoids). In the presence of foreign bodies in the nose, the catarrh is usually unilateral. The disease is generally manifested by persistent coughing, enlarged lymph nodes at the angle of the jaw, oral breathing and other symptoms which usually accompany adenoids (*q. v.*).

Treatment.—As all forms of chronic rhinitis by respiratory interference and secondary glandular infection give rise to more or less impairment of the constitution, the treatment of this condition should embrace local, as well as general, therapeutic measures. The naso-pharynx should be kept clean by antiseptic and oily sprays and the congestion allayed by painting the mucous membrane twice or three times a week with 5 to 10 per cent of argyrol or solargentum, or tannin-glycerine, etc. Excessive hypertrophy should be reduced by trichloracetic acid and similar caustics, and, if these fail, by means of the galvanocautery or nasal scissors. In older children correction of deviated septum.

℞ Thymolis ....gr ii	0.15
Olei eucalypti ....m v	0.3
Albolene ....q. s. ad ℥ ii	60.0
M.	

S.—Nosespray, to be used morning and evening.

Epistaxis

(HEMORRHAGE FROM THE NOSE, NOSEBLEED)

Bleeding from the nose may be due, primarily, to traumatism, external irritation of the mucous membrane from various causes, foreign bodies, etc.; or it may occur as a result of vascular excitement during the course of febrile (typhoid, pneumonia), circulatory (especially after exertion) and pulmonary diseases; and hemorrhagic affections (hemophilia, leukemia). In girls it may occur as vicarious menstruation.

Treatment.—The treatment of epistaxis varies, of course, with the cause. In slight hemorrhage, simple compression of the alæ nasi against the septum acts efficiently. A bland ointment introduced in the nares before the child retires will usually prevent recurrence of the bleeding.

In case of moderate bleeding, sitting posture, head erect, with hands folded over the head, and ice application to the nose and nape of the neck, or instillation of cold water (with some lemon juice, vinegar, alum or potassium permanganate) into the nose will usually suffice. If this fails, the nares should be packed as far back as possible with pledgets of cotton or gauze, dipped in a strong solution of alum, peroxide of hydrogen, or suprarenal gland solution. In secondary epistaxis due to vascular congestion, a small dose of morphine hypo-dermically in conjunction with the aforementioned measures will often act very promptly. As the last resort, we turn to the postnasal tampon, which, as a rule, checks the hemorrhage unless hemophilia is the underlying condition of the bleeding, when the treatment must be directed chiefly against this affection (*q. v.*).

Detection of the local causes is very essential. Every visible bleeding spot should be cauterized with chromic or nitric acid or with the galvanocautery. Constitutional symptoms, if present, should receive prompt attention.

Tumors and Foreign Bodies in the Nose

Mouth breathing, snoring, and nasal speech are not due solely to adenoid vegetations or large tonsils. Not infrequently obstruction to breathing is the result of the presence of mucous polypi (soft, jelly-like), fibrosarcomas (hard and pedunculated), or foreign bodies. The latter are usually beans, pebbles, cherry stones, and so-called rhino-liths. Sooner or later they give rise to a (*unilateral*) foul, bloody discharge and implicate the lacrimal duct and Eustachian canal, and form a reflex cause of persistent irritable cough and asthmatic symptoms. The diagnosis can readily be made by inspection or x-ray examination.

Treatment.—Tumors should be removed with the cold snare, galvanocautery, or by torsion with a slender forceps. Bleeding may be arrested in the manner outlined above.

Foreign bodies if anteriorly situated can readily be removed by air inflation through the free side, or by means of a pointed forceps. If impacted farther back, it is preferable to dislodge the foreign body with a slender hook or forceps under cocaine, and either extract it anteriorly or force it posteriorly into the nasopharynx.

Fig. 69.—Toy ring in antral cavity giving rise to empyema of the antrum of Highmore in a child three years old.

Sinusitis

In children over five years of age, exceptionally in younger ones, we occasionally meet with infections in the accessory sinuses in connection with severe rhinitis, influenza, infected adenoids, etc. In the acute stage the children usually complain of pain at the seat of the

lesion, headache and occasionally dizziness. There is generally also a more or less profuse purulent discharge (unilateral if only one side is affected). In chronic cases the symptoms are usually masked and may be mistaken for those of rhinitis or adenoids. In doubtful cases the Roentgen ray will readily clear up the diagnosis.

These cases are best managed by removal of underlying causes, special attention to cleanliness of the nasopharynx, instillations of argyrol, etc.; if these measures fail, the patient should be intrusted to the care of a rhinologist.

Neglect of sinusitis may not rarely lead to serious consequences (meningitis!).

Pharyngitis Acuta

Acute pharyngitis is rarely primary (streptococcic infection), but quite frequently secondary in nature as a complication of acute rhinitis, tonsillitis, acute exanthematous affections, etc. Primary pharyngitis is ordinarily of short duration and manifested by dryness in the pharynx, pain in swallowing, and moderate rise of temperature. The pharynx is reddened, somewhat swollen, and often granular.

Secondary pharyngitis will be considered in connection with the diseases it complicates.

Treatment.—Attention to the bowels, rest in bed, Priessnitz compresses to the neck and antiseptic sprays to the throat. Liquid non-irritating diet.

Pharyngitis Chronica

It may develop after repeated attacks of acute pharyngitis or as a result of extension of an inflammation from the adjacent structures. The posterior pharyngeal wall not rarely presents a deeply congested granular appearance, and is here and there covered by a tenacious mucous deposit.

The affection is associated with more or less dryness in the throat, hawking and coughing. On examination, the fauces appear swollen and relaxed, the tonsils hypertrophied, and the esophageal opening covered by a thick, grayish-white deposit.

Treatment.—Avoidance and removal of causes. Locally the parts must be kept clean by mild antiseptic sprays (Dobell's solution), and the swelling reduced by nasal instillations of a 5 to 10 per cent solution of argyrol, silvol or solargentum, or by painting the throat with tannin-glycerine (5 per cent). Change of air, iodide of iron, cod liver oil, etc., are very helpful to effect a cure.

ANGINA FOLLICULARIS

ANGINA HERPETIFORMIS, AFTER VESICLE BURST

ANGINA ULCEROSA (VINCENTII)

PLATE V

(Courtesy of Dr. John Zahorsky.)

℞ Supraronal solution (1:2000),
      Dobell's solution ....aa ℥ i | 30.0
      M.
      S.—Throat spray in acute or chronic pharyngitis.

Angina

(Sore Throat)

Tonsillitis Acuta, Amygdalitis, Quinsy

Children under two years of age seem to present a decided immunity against tonsillitis. On the other hand, all forms of angina are extremely common in children over two years old. Those with a "catarrhal habit" are especially prone to contract the disease. Streptococci, staphylococci and pneumococci among other microorganisms, form the most frequent primary cause, and are productive of the usual symptom complex which is characteristic of similar contagious and infectious diseases of childhood. Thus, the attack is ushered in suddenly with a chill, rise of temperature (with evening exacerbations), vomiting (in younger children) and sometimes convulsions. The younger the child the less conspicuous the dysphagia. Hence the importance of a routine examination of the throat in all febrile affections.

To avoid unnecessary repetition, it is advantageous to classify tonsillitis in accordance with the tonsillar deposit as follows:—

1. **Angina Catarrhalis.**—Redness and swelling of one or both faucial tonsils and adjacent tissues. Thin mucous exudation.

2. **Angina Follicularis.**—The deposit begins as one or more white, small pellicles upon the middle or anterior portion of the tonsil. The white dots, at first distinctly isolated, soon coalesce to form yellowish- or greenish-white, elevated patches. These are removable without profuse bleeding, and reform slowly.

3. **Angina Epidemica** (Septic Sore Throat).—The most common appearance is that of follicular tonsillitis, but the constitutional symptoms are much more severe and there is usually marked involvement of the cervical lymph nodes and a tendency to metastatic infection in remote parts of the body. Nausea, vomiting and other gastrointestinal symptoms often predominate. It occurs in epidemics and is usually traceable to infected milk. According to C. H. Dunn, septic angina may be complicated by peritonsillar abscess, suppuration of the cervical lymph nodes, arthritis, peritonitis, pleurisy, pericarditis, pneumonia, laryngitis, endocarditis, phlebitis, nephritis, and septicemia.

4. **Angina Parenchymatosa** (Quinsy, Peritonsillar Abscess).—The tonsil (usually one) and peritonsillar tissue are intensely swollen, often displacing the uvula. It is bluish in color and doughy in consistency. The deposit, at first white, gradually turns yellowish-green, resembling the "point" of an abscess. Pus on puncture.

5. **Angina Herpetiformis.**—The deposit begins with minute vesicles, which tend to burst and leave behind superficial ulcers. This form of amygdalitis usually involves both tonsils and is at times complicated by stomatitis.

6. **Angina Gangrenosa** (Necrotica).—The tonsils are moderately enlarged and almost completely covered by a greenish-yellow, continuous, deposit surrounded by a red zone. The exudation if removed leaves behind a deeply seated ulcer. The deposit often spreads from one tonsil to the other by way of the anterior pillars, palatine arch and uvula.

7. **Angina Ulcerosa** (Vincenti).—It greatly resembles the latter but is usually limited to one tonsil, and occasionally presents a pseudomembrane. It is often associated with stomatitis. Vincent's bacillus in pure culture is almost always found in the exudation.

The course of the different varieties of tonsillitis varies but slightly. After subsidence of the acute initial symptoms previously spoken of, the disease assumes a much milder aspect, except as to prostration, pain on swallowing, and evening exacerbations of the fever. The latter ranges between 102° and 105° F., and is especially high in follicular tonsillitis. More or less marked lymphadenitis is present in all forms of angina, and in accordance with the tonsillar involvement it is either unilateral or bilateral. Parenchymatous angina is not infrequently associated with pseudotorticollis, and pain on moving the jaws is present also in the other forms of the affection.

In uncomplicated cases, recovery is the rule in from three to ten days, but quite a number of deviations from the usual course are observed. Ulcerative angina usually lasts from two to three weeks. Tonsillitis is not rarely the forerunner of true diphtheria or rheumatic affections with their respective complications, and cases are on record where it has proved to be the source of general septic or pyemic infection.

Differential Diagnosis.—Angina may be confounded with influenza, glandular fever, diphtheria and scarlatina. In *influenza* the exudation is slight and not strictly limited to the tonsils; marked adenitis is comparatively rare. Furthermore, influenza is characterized by the simultaneous presence of respiratory, digestive, and often nervous phenomena, while in tonsillitis throat symptoms predominate. *Glandular fever* dif-

fers from tonsillitis by the comparative absence of tonsillar manifestations and preponderance of glandular swelling (also of the bronchial, esophageal and retroperitoneal glands). The distinction between severe cases of tonsillitis and moderately severe forms of diphtheria without a becteriologic examination is often very difficult in the first twenty-four hours of the disease. In pharyngeal *diphtheria* the pseudomembrane appears as a small uneven, grayish white, slightly elevated patch upon the inner tonsillar or faucial surfaces of the throat. The deposit augments by rapid spreading, within a few hours reaching the posterior wall of the pharynx and adjacent structures. The surrounding uncovered areas are grayish in color, due to overcrowding of leucocyte nuclei and mucus beneath. The tonsils are only moderately large in size, but the submaxillary glands are large and hard, assuming the shape of a large walnut, and bulge conspicuously forward. The deposit, if removed, leaves a raw, bleeding surface and rapidly reaccumulates. Diphtheria bacilli are found in the throat. Tonsillitis with and even without erythema may be mistaken for *scarlatina,* and a differential diagnosis is sometimes impossible until a few days after the beginning of the attack.

Treatment.—In view of the possible serious complications, tonsillitis should be arrested at its inception. The following mixture should be used every two hours as a local application, either undiluted, by means of a cotton swab in young children, or diluted .1 to 20 of water, as a gargle, in older ones:

℞	Resorcini ....ℨ ss	2.0
	Acidi carbolici ....gr xx	1.3
	Pulveris camphorae ....gr x	0.6
	Alcoholis ....ℨii	8.0
	Glycerini ....q.s. ad ℥ ii	60.0
	M.	

S.—One teaspoonful in twenty of water as a gargle every two hours, etc.

For the relief of pain, cold Priessnitz's compresses or an ice-collar to the neck, and salicylates internally. The latter is intended also to guard against rheumatic affections. In angina parenchymatosa, if suppuration is inevitable, it should be hastened by hot applications and the abscess opened early. Copious irrigation of the throat with warm boric acid solution is often very efficient. Rest in bed, liquid diet, plenty of water. Avoidance of transmission of the disease. (See "Diphtheria.") Pasteurization of milk whenever tonsillitis appears in epidemic form.

Hypertrophy of the Tonsils

Chronic enlargement of the tonsils often develops after repeated attacks of angina or pharyngitis, not rarely follows scarlatina or diphtheria, and is frequently associated with adenoids. When the tonsils become so large as to obstruct respiration, the same symptom complex makes its gradual appearance as is pathognomonic of adenoids with which it is ordinarily associated. As in the latter anomaly, removal of the hypertrophied tissue is the only actual cure, and unless contraindicated by hemorrhagic diathesis, should be undertaken as early as possible, since the more or less degenerated tumors act not only as a cause of a number of reflex phenomena (*e. g.*, enuresis), but as a harboring place for divers pathogenic bacteria, including the tubercle bacillus. As is well known, rheumatism is frequently traceable to infected tonsils.

Treatment.—Until a few years ago tonsillotomy was looked upon as the operation of choice. Nowadays, however, tonsillectomy, or total enucleation of the tonsils, is generally preferred, especially if the tonsils are submerged.

Tonsillotomy.—This is usually performed in the following manner :—

The patient is placed on a table (if an anesthetic is to be used), or seated on a lap of an assistant or nurse. The arms are immovably fixed by means of a wide towel or sheet. The tonsillotome is introduced into the mouth like a tongue depressor and turned sideways and pressed against the base of the hypertrophied tonsil so that its summit protrudes through the circular opening of the tonsillotome. With the tonsillotome thus fixed and the thumb of the operator in the handle of the blade, the latter is firmly driven through the gland.

The same procedures are repeated for the other tonsil.

Tonsillectomy or Enucleation of Tonsils.—The patient is fully anesthetized, the mouth widely separated with a mouthgag, and the field of operation highly illuminated. The right tonsil is grasped with long but fine tooth forceps, and beginning with the anterior superior portion of the tonsil and pillar, the tonsil is gently loosened from its attachments, by means of a right angled dissecting knife. The enucleated tonsil is then put on a stretch and severed from the adherent constrictor by means of a cold wire snare. The field of operation is kept free from blood and mucus by the suction apparatus. The same procedures are followed for removal of the left tonsil. Some surgeons recommend the application of a tonsil hemostat to prevent sudden hemorrhage. The patient is not allowed to leave the table until the throat is perfectly dry. Tonsillectomy is a more or less capital opera-

tion, and calls for all the precautions, as regards preparation and after treatment, as do other serious operations.

Postoperative Hemorrhage.—Slight bleeding requires no special treatment except ice-collar to the neck. Profuse hemorrhage should be promptly checked by tonsil hemostats, adrenalin and thromboplastin locally, and by all other therapeutic measures generally employed in severe hemorrhage. (See ''Dangers and Accidents Attending Adenoid Operation,'' p. 300.)

Adenoid Vegetations

(Hypertrophy of the Nasopharyngeal or Luschka's Tonsil)

The mucous membrane of the rhinopharynx is normally rich in lymphoid or adenoid tissue which bears the name of nasopharyngeal or Luschka's tonsil. Like the faucial tonsils, the latter is subject to fre-

Fig. 70.—Adenoids in a boy eleven years old. Note characteristic, dull, facial features and contracted chest.

quent attacks of inflammation with secondary hypertrophy. Whenever the hypertrophied adenoid tissue assumes such proportions as to more or less fill the nasopharyngeal space and obstruct nasal breathing, a pathognomonic clinical syndrome develops which, though apparently insignificant in its lesion, is often very verious in its immediate and remote consequences.

The clinical picture unfolds gradually, almost insidiously, growing more pronounced from time to time as the patient "catches cold." The child is unable to clear the nasopharynx, and the retained irritating nasal discharge helps to swell the adenoid tissue and to obstruct the rhinopharynx. He is thus forced to breathe through the mouth. As immediate results, we find that he constantly keeps his mouth open, especially during sleep, which is greatly disturbed, and, as a rule, he snores heavily. As the nasal obstruction increases, he is frequently awakened by extreme dryness of the throat, and a croupy harassing

Fig. 71.—Spinal curvature (stooping) secondary to adenoids.

cough. In the morning he is tired, complains of headache, is drowsy and apathetic. His speech is dull, nasal (*m* and *n* sound like *b* and *d*), hesitating, and sometimes stuttering.*

If it were possible to bring these little sufferers under proper treatment at this stage of the disease, quick and uneventful recovery would be the rule. Unfortunately, however, the laity, nay, the physicians as well, rarely think these symptoms of sufficient gravity to necessitate

*It should be remembered, however, that the presence of adenoids does not necessarily produce the typical symptoms of the disease. It all depends upon the proportionate size of the tumor to that of rhinopharynx.

medical and particularly surgical intervention. The deplorable condition is therefore allowed to proceed and the tumor to spread and sprout. The sequelæ appear in rapid succession. The labored breathing sooner or later produces deformity of the thorax (pigeon breast) and often curvature of the spine. Owing to nonparticipation of the nose in respiration there is gradual atrophy of the levators alæ nasi et labii superiores, the depressors alæ nasi, and the septum mobile. The nose becomes pinched and pointed, the external angle of the eye deeper than the internal, the lower lid droops, the lower jaw sinks down, and the face assumes that dull, fixed and irresolute expression which is so characteristic of adenoids. In addition to this, hearing is impaired as a result of secondary catarrhal inflammation of the Eustachian tube, etc. The child is absent-minded and dull of perception, does poorly at school, and becomes the target for abuse and corporal punishment by teachers and parents—all for no fault of his. When brought to the physician— often chiefly on account of impaired hearing—the diagnosis can readily be made by mere inspection. Such a superficial examination, however, should not be relied on, as similar symptoms are produced by nasal obstruction from other causes (deformities, growths, foreign bodies, etc.). Inspection of the mouth reveals the bony palate high and narrow, leaving insufficient space for the teeth and causing their displacement. The faucial tonsils are greatly enlarged (in about 25 per cent of the cases), the posterior pharyngeal wall is granular, and, with the velum palati raised, often shows the distal ends of the adenoid vegetation. Rhinoscopy confirms the presence in the nasopharyngeal space of a palered, smooth, soft tumor which sometimes resembles a mass of earthworms. It bleeds readily. The diagnosis is further corroborated by palpating with the finger the soft masses blocking the rhinopharynx, or by nipping off a small portion of the adenoid vegetations by means of adenoid forceps introduced behind the velum palati.

Treatment.—The diagnosis once established, the treatment should be prompt and energetic. *Mild cases in their early stages* may be arrested at their inception by scrupulous cleanliness of the nasopharynx, local applications of Lugol's solution or 2 per cent nitrate of silver, or 5 to 10 per cent argyrol, silvol or solargentum, change of air, outdoor exercise, cold shower baths, and hematinics and alteratives internally. These procedures should also be followed in cases with *hemorrhagic diathesis* where an operation is contraindicated for fear of uncontrollable bleeding, and in those associated with other grave affections, *e. g.*, acute or subacute endocarditis. In all other cases, removal of the adenoids is the only actual cure, and should be undertaken as early as possible. The mode of procedure varies with each individual case. In young

children under three years of age, the operation may be performed without (preferably with!) an anesthetic, in the sitting posture; in older ones or in those who are hypersensitive to pain and shock, under primary anesthesia with ether (drop by drop method), ethyl chloride or bromide or nitrous oxide gas, in the recumbent posture. The child's arms are fastened to the sides of the thorax by a wide towel, and his jaws are separated by a mouth gag placed between the left upper and lower teeth. The operator stands on the right side of the patient and introduces the adenoid curette sideways into the latter's mouth and passes it beneath the soft palate and up along the anterior wall until he reaches the vault of the rhinopharynx. The physician then implants the cutting edge of the instrument into the adenoid mass and makes a firm semicircular movement, directed backward, downward and forward. One such movement usually suffices to remove the tumor. It may be followed, however, by a few light, similar strokes, to smoothen the rough edges. The patient is then turned on the side to allow the blood to drain into a basin. This may be facilitated by the injection of ice-cold water through the nostrils. After arresting the more or less profuse hemorrhage, which always accompanies the operation, the child is put to bed for a few hours until he has regained full consciousness and is kept indoors for a day or two on a nonirritating, cool, liquid diet.

After-Treatment.—To prevent the recurrence of the adenoids, which is prone to take place in children with a tendency toward glandular hyperplasia, it is advantageous to instill into each nostril a few drops of Lugol's solution, once every other day for a period of about four weeks, and to use an oily antiseptic spray for several weeks thereafter. This procedure will prevent also adhesions between the cut surfaces and the soft palate. Delicate children should be put on syrup of the iodide of iron, cod liver oil, etc. To regulate nasal breathing, it is often necessary by means of a bandage to keep the mouth closed, especially at night, and to have the child take prolonged breathing exercises with closed mouth. Impaired speech sometimes calls for instruction in speaking or, in the event of a paretic condition of the velum palati arising from inactivity, for treatment by electricity and tonics. In the majority of instances, however, the operation is followed by immediate *restitutio ad integrum*. All reflex symptoms and, to a great extent, even the deformities of the thorax subside rapidly.

Dangers and Accidents Attending Adenoid and Tonsil Operations

Simple and harmless as the operation is under ordinary conditions, it is not always free from danger. As in more serious operations, the possibility of fatality from the effect of the anesthetic or infection is

gravely to be borne in mind, and the frequency of primary or secondary —occasionally fatal—hemorrhage should engage the constant attention of the operator. Hence the importance also of testing the coagulability of the patient's blood before the operation.

To obviate untoward complications all such preparations should be made as are customary with capital operative work. Ethyl chloride and ether (drop by drop method) should be the anesthetic of choice, and primary in preference to deep anesthesia. The instruments to be used should be carefully sterilized, and the field of operation and everything coming in contact with it rendered as aseptic as possible. Before beginning the operation, the surgeon should test the efficiency and entirety of his instruments, and see to it that he is amply supplied with all such drugs (peroxide of hydrogen, suprarenal gland in solution 1:1000, thromboplastin, the tincture of chloride of iron, etc.), and with implements (postnasal tampon, artery forceps, sponge holder and styptic gauze—which can be used to exert direct pressure upon the bleeding spot; actual cautery, etc.), as will enable him promptly to check profuse hemorrhage. Postoperative fever is generally due to some form of throat infection and hence should promptly be treated by local application of tincture of iodine or argyrol (25 per cent solution).

Retropharyngeal Abscess

(RETROPHARYNGEAL LYMPHADENITIS)

Retropharyngeal abscess is a disease of early infancy and childhood when the retropharyngeal lymph nodes are in a state of highest development. It usually begins as retropharyngeal lymphadenitis, most frequently the result of infection by offensive nasopharyngeal discharges. More rarely it is due to spondylitis of the cervical vertebræ, or occurs as a metastatic abscess, or in consequence of trauma. Not all cases of lymphadenitis undergo suppuration; on the contrary, quite a number retrogress and escape attention. Hence the apparent rarity of retropharyngeal disease. Some cases undergo suppuration and break spontaneously, and others run a rather latent course, and when seen by the physician present fully developed abscesses. Digital examination of the throat usually reveals, at a late stage, a round or oval fluctuating mass the size of a pigeon's egg, in the median line of the pharynx, and more rarely, laterally on a line with the velum palati or somewhat below it. In the more advanced stages the abscess may be recognized as a bluish-red tumor on ordinary inspection of the pharynx.

The symptoms vary with the size of the tumor. In marked cases they consist of dysphagia, snoring respiration, especially during sleep, muffled

voice and with progressive growth of the swelling, dyspnea and attacks of asphyxia. Where deglutition is very painful there is also sympathetic pseudotorticollis. Occasionally the submaxillary, parotid and other neighboring glands are involved; and in spontaneous rupture of the abscess metastatic abscesses are apt to develop in the ear and the supraclavicular fossa, mediastinum, and lungs. The temperature is usually high in the early stage of the disease, and remittent later.

Treatment.—Early opening of the abscess is therefore imperative. This is best accomplished by gently perforating it by means of a pointed artery clamp and widening the puncture by opening the clamp.

Fig. 72.—Retropharyngeal abscess in a ten-month-old infant. Note characteristic attitude of mouth, head and neck.

Before opening the abscess the child's head is held upright and steadied from behind by an assistant. As soon as the perforation is made, the child's head should be promptly bent forward to prevent the pus from entering the larynx (danger of asphyxia, aspiration pneumonia, etc.) and the nose and throat cleared of blood, pus and mucus.

In multiple communicating abscesses with palpable involvement of the adjacent gland, the operation is preferably performed (with a knife) from the outside, so as to afford thorough drainage. Sometimes it is of advantage to poultice the abscess for a few days before opening it.

Relief from the symptoms is very prompt after evacuation of the pus. Rapid recovery, however, occurs only in primary streptococcic or staphylococcic abscesses. In metastatic and tuberculous abscesses (especially the latter) the disease proceeds a protracted course, the prognosis depending upon the original disease and the age and vitality of the patient. General attention to the nasopharynx. Hematinics.

Otitis Media

(Otitis Externa, Furunculosis, Foreign Bodies in the Ear, Mastoiditis)

The gravest feature of nasopharyngeal affections, be they primary or secondary, is their great tendency to ear complications. The nasopharynx and ear being in direct communication through the Eustachian tube, infectious material can readily travel from the nose and throat to the middle ear and transfer the disease from one locality to the other. Hence the frequency of ear disease in rhinitis, adenoids, divers exanthematous affections, influenza, etc. Only a small percentage of cases of otitis media are contracted through traumatism, sea bathing, or extension of an inflammation from the external auditory meatus; and, in infants, middle-ear disease with masked symptoms is occasionally observed in connection with wasting diseases (e. g., tuberculosis, marasmus, syphilis). Epidemics of ear disease are not rare.

The infection may remain limited to the Eustachian tube (*catarrh of the Eustachian canal*), and give rise to very few and mild symptoms. The child may complain of earache for a day or two, perhaps, wake up at night with a crying spell, but get immediate and usually permanent relief after application of heat or some "ear drops." Sometimes the pain may return and get much more intense, and examination of the drum would show injection of the drum or, perhaps, a slight mucopurulent discharge indicating spontaneous rupture of the membrane. The discharge may continue for a few days or weeks and disappear without further ado. In another group of cases, due to greater virulence of the infective material or, possibly, neglect, the inflammatory process pursues a more violent course (*otitis media purulenta*). The temperature rises, the earache is very intense, (but may be absent!), the child is very restless, cries almost incessantly, rubs or strikes the ear with its hands, and, as the symptoms persist, there may be vomiting and cerebral irritation up to convulsions. If the pus is not evacuated, we soon find that it eats its way into the deeper structures, leading either to an acute or chronic involvement of the bone (*mastoiditis*). In severe infections this stage of the disease is often reached within a few days. The

aforementioned constitutional symptoms are greatly exaggerated. The local signs—in addition to intense earache, deafness, headache and marked congestion of the drum—are also augmented by tenderness over the mastoid process and by edema of the tissues covering the bone, extending downward along the entire side of the neck and backward to the retromaxillary fossa, pushing the auricle forward. The upper and lower walls of the meatus are more or less swollen and the drum is highly inflamed, bulging and irregular in contour, while the posterior superior quadrant of the drum with the adjacent wall of the canal is sagging. The further course of the affection depends greatly upon the mode of treatment. If the inflammatory process is allowed to continue, the pus may find its way either externally, somewhere along the side of the neck, into the throat (retropharyngeal abscess), or, in malignant cases, into the lateral sinus (*phlebitis, thrombosis*) or the middle fossa of the skull (*meningitis, purulent encephalitis*). The same grave condition is sometimes observed in otitis pursuing a very slow course—months or years. In these cases it is usually found that the patient is suffering from recurrent attacks of earache with or without profuse purulent discharge, more or less severe headache, dizziness, occasional rise of temperature, tenderness over the mastoid process, and, toward the end, loss of weight, anorexia, persistent headache and repeated vomiting.

The disease having reached this deplorable stage, one is very rarely apt to err in the diagnosis. A question may arise as to whether the meningeal symptoms are secondary to otitis or to some other affection (*e. g.*, pneumonia, sepsis), or primary in character. A history of ear disease and the presence of local ear symptoms (discharge, inflammation of the drum, etc.) at once point to its true nature. Nor is there any difficulty in diagnosing otitis media purulenta with acute symptoms. The diagnosis, however, is not so easy in cases with an insidious course. It is especially difficult when the ear symptoms are masked by manifestations of the primary affection (*e. g.*, influenza), but an electro-otoscopic examination almost invariably clears up the diagnosis, and should always be resorted to whenever inexplicable pain or temperature prevails. In fact, no examination of a baby is complete without such an examination. Only very recently I had occasion to find double otitis in a boy fourteen months old who, for three weeks, was treated by a prominent clinician for ''central pneumonia.'' Mild cases of middle ear disease may be mistaken for *otitis externa*. In this affection, however, the local signs are limited to the external auditory canal (redness and narrowing of the meatus without involvement of the drum). Similarly, middle ear disease may be confounded with *furunculosis* or *foreign bodies* in the auditory meatus, but these can readily be eliminated by

an otoscopic examination showing the seat of the lesion. Occasionally an abscess in the external canal burrowing itself through the cartilaginous portion of the canal in back of the ear may be mistaken for mastoid abscess; in such cases constitutional symptoms and inflammation of the drum are absent and the abscess is superficial and communicating with the swelling in the external canal.

Treatment.—Bearing in mind the great tendency of nasopharyngeal affections to lead to ear disease, and the latter to become a source of everlasting misery and death, it is self-evident that all precautions should be taken to prevent the causes and their dreadful results. During the course of acute febrile, especially exanthematous diseases, the nasopharynx should receive especial attention in the way of careful, gentle cleansing. Warm salt water or albolene, or some silver preparation should be instilled into the nose twice daily, preferably with a spoon or dropper, lest forcible syringing may drive the discharge from the nasopharynx into the Eustachian tube. Hypertrophied tonsils and adenoids should be removed (during the quiescent stage of the otitis!) and chronic nasopharyngeal catarrh treated with appropriate remedies. The instillations should also be continued after the appearance of ear symptoms, and as long as the membrane is intact syringing of the ear with warm boracic acid solution will prove beneficial. If the otitis continues and the drum does not rupture spontaneously, free paracentesis, preferably under primary anesthesia, should be performed without delay, to allow the pus to escape. The mode of after-treatment is still subject to controvery, several prominent otologists preferring the "dry" method (drying of the external auditory canal several times a day and loosely draining with absorbent gauze) to repeated syringing. Some otologists recommend to cleanse the ear once a day by suction and to follow it up with packing. Where the discharge continues, instillation of a few drops of a 2 per cent solution of nitrate of silver, or in very chronic cases cauterization of the tympanum with trichloracetic acid will be found to act splendidly. If sensitiveness over the mastoid is detected and the constitutional symptoms show that the disease is rapidly growing worse, an attempt should be made to arrest its progress by a new paracentesis, ice bags and leeches and, if improvement does not set in early, there is nothing else left but immediately to proceed with opening of the mastoid process with a chisel to prevent the pus invading the sinus, meninges or brain substance. In the majority of instances a radical mastoid operation is a life-saving procedure. Unfortunately, this operation is not rarely undertaken either too late or on a patient in a state of very low vitality from the baneful effect of the primary

disease, so that the results are not always very gratifying. It is questionable whether operative interference is to be advised after the disease has spread to the meninges or brain. The recoveries in these cases are certainly very few and far between.

Pain should be relieved by small doses of codeine, and other symptoms arising should be met in accordance with indications. Change of air often works wonders in recalcitrant cases.

℞	Acidi Phenolis	gr v	0.333
	Mentholis	gr ii	0.133
	Glycerini	℥ iv	15.00
	M.		
	S.—"Ear drops," in acute cases.		
℞	Hydrargyri Chloridi Corrossivi	gr ss	0.003
	Alcoholis	℥ iv	15.00
	M.		
	S.—"Ear drops," in chronic cases.		

Deafness

From a study of 1,076 congenitally deaf children, Yearsley reports heredity and consanguinity to be the most important factors; with alcoholism, insanity, and syphilis the most important minor causes. Of 592 cases of acquired deaf-mutism 72.2 per cent were due to suppurative or catarrhal middle ear disease, in which infectious fevers and adenoids played an important part. Statistics collected by other clinicians show similar data regarding the etiologic factors of deafness.

Testing the Hearing.—The acuteness of hearing is determined by the watch test and the voice test applied separately to each ear. During both the watch and the voice tests, the eyes of the patient should be closed, in order that lip reading may not be possible, and that the distance of the watch or the speaker may not influence the patient. In tests relative to differential diagnosis and prognosis the tuning fork is used.

Weber's Test.—In this test a C_2 tuning fork, having 512 vibrations per second, is vibrated and the handle placed against the upper central incisor teeth or upon the middle line of the cranium. If the sound is heard better in the afflicted ear it is indicative of some affection of the conducting apparatus, such as middle ear disease, impacted cerumen in the external auditory canal, or occlusion of the Eustachian tube; while if heard better in the normal or less afflicted ear, it is the perceptive, or nerve, apparatus that is at fault.

Rinné's Test.—This test depends upon the fact that the normal ear

is more sensitive to sounds transmitted through the air than to those transmitted by the bony framework of the ear. In a normal ear, if the handle of a vibrating C_2 (512 vibrations) tuning fork is held against the mastoid until the patient no longer hears any sound, and then the free tips of the fork be brought close to the external ear, the sound will be heard again. This is known as *positive Rinné*. If, however, the sound is not heard again when the fork is thus transposed, it is known as *negative Rinné*. In a defective ear, a negative Rinné test shows a relative reduction of aerial conduction or a similar increase in bone conduction and indicates obstruction or disease of the normal conducting apparatus; while a positive Rinné test, in a defective ear, is an indication of a lesion in the perceptive apparatus of the internal ear.

Schwabach's Test.—This test depends upon the fact that in middle ear disease, a fork vibrating in contact with the cranium is heard longer in an affected than in a normal ear. When the auditory nerve is affected, it is heard longer by the normal ear. The fork is struck and placed on the patient's mastoid and when the patient ceases to perceive the sound, the fork is transferred to the examiner's mastoid. If still heard by the normal ear of the examiner, it indicates labyrinthine disease in the patient. If not heard, the test is reversed, the examiner first placing the vibrating fork on his own mastoid, and, when the sound has died away, transfers it at once to the patient's. If heard by the patient after the examiner's normal ear has ceased to hear it, an obstruction of sound condition, but not disease of the nerve, is indicated.

Interpretation of Above Tests

A negative Rinné test indicates middle ear disease which should be partly or entirely benefited by treatment. A positive Rinné test indicates nerve deafness which, with a few exceptions, implies an unfavorable prognosis. The Schwabach test deduces an abnormal condition of the conducting apparatus (middle ear) when bone conduction is lengthened and, conversely, disturbance of the nervous mechanism when bone conduction is impaired. The Weber test is only valuable in indicating disease of the perceptive apparatus in unilateral deafness, when the sound is accentuated in the normal ear; the chief objection to the test being the uncertainty of the localization of the auditory perception in one ear. Tuning-fork tests are said to be of value as aids to prognosis when they point to middle ear disease. However, the deductions drawn from these tests should be used with caution as the pitch

and intensity of the sounds employed may sometimes cause them to vary and even to be the direct opposite of what the formulated rules would lead us to expect. Even with indications of a diseased perceptive apparatus, supported by many tests, an unfavorable prognosis should be withheld until treatment has been administered and found unavailing.

Indications of Labyrinth or Auditory Nerve Deafness

1. When the tuning fork is heard better through the air.
2. When the power of hearing is better in a quiet place.
3. When noises are markedly annoying.
4. When inflation of the middle ear makes the hearing worse.

Among the various tests employed in the diagnosis of labyrinthine disease that of Bárány is without danger and may prove of service. According to Bárány, when the vestibule is healthy, the injection of water at a temperature of, say 80° F. into the external auditory canal will develop a circular nystagmus toward the opposite side. On the other hand, if the temperature of the water be above that of the body, the nystagmus will be toward the ear syringed, while if the labyrinth be destroyed no nystagmus will develop.

Treatment.—Careful attention to existing external and middle ear diseases. Tonics and electric vibration may prove beneficial in labyrinth deafness. Iodides and mercury are always worth trying even if the Wassermann test is negative.

Laryngitis Acuta

(CATARRHAL LARYNGITIS, SPASMODIC OR FALSE CROUP, LARYNGITIS STRIDULA, MEMBRANOUS, NONDIPHTHERITIC CROUP)

Acute primary, idiopathic laryngitis is comparatively rare in children, except as the result of the traumatic action of strong gases, vapors, fluids or excessive heat. On the other hand, laryngitis quite frequently occurs in conjunction with divers acute exanthematous diseases, especially measles and influenza, often follows attacks of rhinitis, pharyngitis, tonsillitis and esophagitis, and may develop in connection with intra- and extra-laryngeal growths. This so-called secondary laryngitis affects children principally of from two to ten years of age.

The severity of the symptoms is often by far out of proportion to that of the underlying anatomic lesion. Thus, simple hyperemia of

only a small portion of the laryngeal mucous membrane not rarely gives rise to marked symptoms of suffocation.

Several forms of laryngitis are noted in practice:—

1. **Catarrhal Laryngitis.**—The child complains of sore throat and sensitiveness of the larynx to pressure. The cough is dry, short, and barking; the voice husky or only slightly muffled. Respiration is normal; fever is absent or slight. Expectoration is at first slight and of a mucous nature, later more profuse and mucopurulent. The attack lasts about a week.

Occasionally, especially in neglected cases or in those suffering from affections of the nasopharynx, the laryngitis may pursue a chronic course with a tendency to permanent alteration of the voice. In this event laryngoscopic examination usually reveals a moderate hyperemia of the laryngeal mucous membrane, and in some cases slight erosions.

2. **Spasmodic Laryngitis (Laryngitis Stridula, False Croup).**—It develops, either very suddenly or after a few days' illness, with catarrhal laryngitis or nasopharyngitis. Sudden attacks usually occur in children under eight years of age, more frequently in boys than in girls. After retiring apparently healthy and sleeping fairly well until about midnight (this may also happen during the day after prolonged sleep, when the nasopharyngeal or laryngeal secretion desiccates and gives rise to irritation of the larynx, and possibly edema of the subchordal tissue) the child wakes up with a harsh, croupy cough, interrupted by deep inspiratory stridor. The child looks frightened and anxiously gasps for air, its face is flushed and bathed in perspiration, its eyes stare and its lips are cyanosed, and the whole clinical picture is very alarming. The dyspnea usually passes off in a few minutes, but may last hours with slight remissions and gradual improvement. Ordinarily the child is well again in the morning except for a simple mild laryngitis which may subside in two to ten days or give rise to renewals of the attacks for a few successive nights. Sometimes the paroxysm may be so severe as to require intubation or tracheotomy for immediate relief. Spasmodic croup occasionally forms the beginning of pertussis, measles, influenza or membranous, nondiphtheritic croup. It should not be mistaken for spasmus glottidis (q. v.).

3. **Membranous, Nondiphtheritic Laryngitis.**—In the beginning the symptoms are those of simple laryngitis. Very soon, however, the catarrh is increased in intensity. The cough becomes harsher and more croupy, the voice hoarse (sometimes aphonia), inspiration prolonged and expiration noisy. It may begin also with bronchial catarrh and

become suddenly complicated by fibrinous tracheolaryngitis—*ascending croup*—reach a very high degree of intensity, become more severe from hour to hour, and threaten suffocation, if not immediately relieved by intubation or tracheotomy. The aspect is still worse when the croupous inflammation descends into the bronchi—*bronchial croup*. In this condition the patient may cough up white reticulated shreds (which float in water) or complete cylinders with dichotomic ramifications or multiple dendritic branchings. The prognosis in such cases is very grave. The pulse fails, the dyspnea and cyanosis increase, the patients fall into a state of sopor and die from collapse. Not infrequently fatal brain symptoms occur as a result of passive venous congestion in the brain and transudation in the ventricles. The course and termination of the disease, however, is not always so bad, and quite a number of uncomplicated (sometimes complicated by bronchopneumonia) cases recover without much ado.

This nondiphtheritic form of laryngitis is often mistaken for diphtheritic membranous laryngitis, but a diagnosis can in the majority of cases be made with the aid of the following differential points:

MEMBRANOUS DIPHTHERITIC LARYNGITIS	MEMBRANOUS NONDIPHTHERITIC LARYNGITIS
Diphtheria bacilli present.	Absent. Streptococci, Staphylococci or Pneumococci present.
Distinctly contagious, giving also a history of contagion.	Not contagious.
Early enlargement of the submaxillary glands.	Submaxillary glands, as a rule, not involved or slightly so.
Diphtheritic patches are found, as a rule, on the fauces and posterior pharyngeal wall.	The fauces may be covered with a mucous exudation, which can easily be wiped off.
Albuminuria usually present.	Absent.

Treatment.—Mild cases do nicely on very simple therapeutic measures, such as rest in bed, attention to the nasopharynx (instillations of warm boracic acid solutions several times daily; occasionally also 5 per cent argyrol or silvol), hot baths, hot drinks (tea, lemonade, milk and seltzer, Priessnitz's compresses or turpentine and camphorated oil to the neck and a few doses of sodium salicylate internally to relieve the sore throat and to stimulate diaphoresis.

Should there be any tendency for desiccation of the laryngeal secretion, softening of the same should be endeavored by means of expectorants, steam inhalations and emetics. In the majority of instances this mode of treatment prevents the occurrence of attacks of spasmodic laryngitis.

℞ Vini ipecachuanhæ ....℥ ss | 2.00
   Syr. scillæ comp ....℥ i | 4.00
   Syr. senegæ ....℥ ii | 8.00
   Codeinae sulph. ....gr ss | 0.033
   Ext. glycyrrhizæ fl. ....℥ ii | 8.00
   Aquae ....q.s. ad ℥ ii | 60.00
   M.

S.—One teaspoonful every two to four hours for a child 3 years old.

℞ Eucalyptol ....℥ i | 4.00
   Tinct. benzoini comp. ....℥ ii | 60.00
   M.

S.—One teaspoonful in a pint of hot water for inhalation.

Sudden paroxysms of false croup are best remedied by ice collar, prompt emesis, a hot mustard bath (see p. 92), a large dose of sodium bromide, a hypodermatic injection of morphine 1/20 grain and atropine 1/400 grain, counterirritation by a strong sinapism and, if the cyanosis increases notwithstanding, intubation or tracheotomy.

The management of membranous nondiphtheritic croup is frequently quite a difficult proposition. Hence, the importance of its prevention by early attention to catarrhal laryngitis. Steam inhalation and emesis are useful remedies, and inhalation of a few drops of chloroform is often effective to relieve threatening dyspnea. Severe cases call for early intubation or tracheotomy. Recurrent laryngeal spasm sometimes yields to spraying of the larynx with 2 per cent solution of cocaine. As diphtheria antitoxin carefully administered is a safe remedy, it is always advisable to resort to it, although bacteriologic examination of the pseudomembrane fails to reveal the diphtheria bacillus. Mixed antistreptococcic, staphylococcic and pneumococcic sera are also deserving of trial.

Prophylaxis.—Removal of local causes, such as adenoids and large tonsils; change of air; tonics, especially cod liver oil.

Laryngitis Chronica

Chronic laryngitis may follow repeated attacks of acute catarrhal or diphtheritic laryngitis or develop slowly by extension of inflammation from the neighboring structures. Overexertion of the voice and excessive smoking in boys are occasional causes.

Laryngoscopic examination shows hyperemia and swelling of the mucous membrane of the larynx which vary in extent with the duration of the affection. The mucous membrane is sometimes covered with granulations, and in severe cases shows more or less superficial ulceration. There is a moderate secretion of mucus and pus which has

a tendency to desiccate, and gives the sensation of a foreign body in the throat. The cough is usually insignificant; occasionally, however, troublesome, harsh and barking, especially at night.

Diagnosis.—Although syphilis and tuberculosis of the throat are comparatively rare in children, their presence should always be suspected and looked for in obstinate laryngitis. The following differential points are helpful in the diagnosis:

	SIMPLE LARYNGITIS	SYPHILITIC		TUBERCULOUS
		SECONDARY	TERTIARY	
Lesion	Hyperemia, slight thickening, erosion of mucous membrane, rarely slight ulceration.	Mottled hyperemia, superficial ulceration.	Deep, angry ulcers, cicatrices, stenosis.	Anemia, grayish color, solid thickening, worm-eaten ulcers.
Expectoration	Free from tubercle bacilli.	Spirochetes.	The same.	Bacilli present.
Deglutition ..	Usually painless.	Normal.	Difficult.	Very painful.
Cough	Dry or moist, painless.	Slight hacking.	Infrequent.	Severe, as a rule.
Respiration ..	Normal.	Unaltered.	Embarrassed with stenosis.	Early acceleration.
Voice	Variable.	Hoarse, nasal.	Raucous, husky.	Partial or complete aphonia.
Complications	Nasopharynx; general health unaffected.	Syphilitic lesions elsewhere.	The same.	Involvement of lungs, emaciation.

Treatment.—Attention to existing causes, especially adenoids and enlarged tonsils, if present; local application, three times a week, of nitrate of silver (1 per cent to 2 per cent), glycerate of tannin (10 per cent), or chloride of zinc (2 per cent to 4 per cent); steam inhalation (see p. 311); cleansing of the nose and throat, three times a day, with Dobell's solution, and the like, will very promptly effect a cure, provided the laryngeal affection is not based on some grave constitutional affection, or benign (papilloma) or malignant growths. Rest to the voice is of material benefit. In very protracted cases change of air and constitutional treatment. Faradization of the larynx is often very serviceable to relieve aphonia.

℞	Codeinæ sulph.	gr ss	0.033
	Creosoti carbon.	ʒi	4.00
	Syr. acaciæ	q.s ad ℥ii	60.00
	M.		
	S.—One teaspoonful every three hours for a child six years old.		

Edema Glottidis

(Submucous Laryngitis, Phlegmonous Laryngitis)

Edema of the upper portion of the larynx occurs in two forms: Active (inflammatory, phlegmonous), and passive (serous). Inflammatory edema may be primary, usually traumatic (*e. g.*, scalds or burns), or secondary, as a result of extension of inflammation from neighboring structures. Passive edema is usually observed in connection with grave kidney and heart disease—often long before dropsy is manifested in any other part of the body—and secondarily to pressure on the larynx by swellings or growths.

Pathologically edema of the larynx consists of a yellowish-white or reddish tumefaction—a serous, seropurulent or sanguinolent transudation into the submucosa—involving the upper portions of the larynx, the epiglottis, the aryepiglottic folds, the false (rarely the true) vocal cords, and the mucous membrane of the arytenoid cartilages.

These local changes can readily be detected by inspection of the larynx, often without the mirror, by simply depressing the tongue and pulling it forward, and by digital examination.

The result of such swelling of the laryngeal tissues is quite obvious—namely, interference with normal respiration. The dyspnea is at first paroxysmal, and, if the edema is not very marked, only moderately severe. The poor little patient hacks and coughs, in vain trying to clear the throat. If the edema advances, which is apt to occur in severe traumatic cases, the dyspnea may become extreme, and symptoms of asphyxia may set in which, if not promptly relieved, may lead to a fatal issue.

Edema glottidis should not be mistaken for spasmodic croup or asthma!

Treatment.—Partial edema may be reduced by ice bags to the neck, swallowing of ice, local application of suprarenal extract solution (1:1000) and morphine and pilocarpine hypodermically. In severe cases, scarification and, if need be, tracheotomy should be resorted to in addition to the mode of treatment just outlined. Recurrence of an attack of passive edema should be prevented by prompt attention to the etiologic factors.

Laryngeal Tumors

Neoplasms of the larynx are very rarely seen in children. This is especially true of malignant growths. Granulomata are occasionally observed after tracheotomy. *Papillomata* are not quite so rare, and are sometimes congenital, in which event the symptoms usually appear soon

after birth. Their usual seat is at the true vocal cords, and if of considerable size they give rise to obstinate cough, hoarseness, dyspnea and attacks of asphyxia. These symptoms develop, however, gradually, and sometimes disappear spontaneously owing to retrograde metamorphosis of the tumor. Recurrences after removal of the tumor are frequent. Laryngeal neoplasms may be confounded with adenoids, retropharyngeal abscess and croup, but the diagnosis can readily be made by laryngoscopic examination. Operative treatment should be instituted only in cases presenting troublesome symptoms. Endolaryngeal removal of the growth is the procedure of choice. Tracheotomy is indispensable in threatening asphyxia.

Foreign Bodies in the Larynx

Various articles of food, little playthings, buttons, needles, ascarides, etc., may find their way into the larynx. Small foreign bodies are usually expelled by the attacks of forcible coughing. Large nonimpacted articles may be removed by an extubator or similar forceps after cocainizing the upper part of the larynx. Foreign bodies firmly impacted in the larynx should be removed under anesthesia through the tracheotomy incision. In threatening asphyxia, tracheotomy should be performed immediately irrespective of subsequent procedures. To reduce hyperemia, ice externally and internally. Local antiphlogosis (Lugol's solution, 1 per cent nitrate of silver) after removal of the foreign body.

Anodynes for the relief of pain and irritability. (For removal of ascarides see p. 280.)

Diseases of the Bronchial Tubes, Lungs and Pleura

Bronchitis Acuta

(TRACHEOBRONCHITIS, FIBRINOUS BRONCHITIS, CAPILLARY BRONCHITIS)

As the term indicates *tracheobronchitis* is a catarrhal inflammation of the trachea and large bronchi. It usually develops, by extension, secondarily to nasopharyngeal and laryngeal catarrh, either in association with ordinary colds or in consequence of specific infections such as influenza, pertussis, diphtheria and the like. Occasionally it is met as a result of traumatism by irritating vapors or dust. Except for the harsh cough, which is at first dry and later soft and yielding, a moderate amount of mucous and mucopurulent expectoration, slight embarrassment of respiration, slight temperature and anorexia, simple bronchitis is usually a benign affection terminating favorably within a week or ten days. Its seriousness consists only in its tendency towards the develop-

ment in bronchopneumonia—which is most apt to occur in young infants or older children whose health has been undermined by previous illness. The physical signs are usually limited to diffuse large soft râles which temporarily disappear after brisk coughing.

Fibrinous Bronchitis.—This form differs from simple bronchitis by the presence of membranous masses of mucus and fibrin in the expectoration, in the form of bronchial casts. The casts correspond to the size and depth of the bronchi involved. Until relieved by the ejection of the casts, the patients suffer from more or less marked dyspnea and fever.

Capillary Bronchitis.—In this form of the disease the small bronchi, the bronchioles, are involved, and it is often questionable whether or not the inflammation actually remains limited to the fine bronchi or extends to the pulmonary alveoli. As a rule, capillary bronchitis begins as a simple bronchitis, but as it progresses, its symptomatology is essentially the same as in the early stages of bronchopneumonia: thus, painful cough, more or less dyspnea, moderate or high fever, often vomiting and twitching, pallor, and cyanosis. Fine sibilant râles are heard over different portions of the chest, and sometimes also fine crepitation. Unless the affection is arrested in its early course, its transition into bronchopneumonia is the rule.

Treatment.—The patient should be kept in bed in a well-ventilated warm room; the diet reduced to liquids, and the bowels regulated. The nasopharynx should be cleansed a few times daily with Dobell's solution (50 per cent) or weak solutions of the newer silver preparations. Inhalations of antiseptic vapors (with the compound tincture of benzoin and eucalyptol) may be added as a routine procedure. Where the cough is painful, and distressing, the flaxseed mustard poultice recommended in bronchopneumonia (*q. v.*) will often give relief and occasionally arrest the disease in its inception. The following preparations will be found very serviceable:

℞	Liq. Ammonii Anisati		
	Vini Ipecacuanhæ	aa ʒ ss	2.0
	Potassii Citratis	ʒ i	4.0
	Syrupi Picis	aa	
	Glycerini	ʒ iv	15.0
	Aquæ Anisi	q.s. ad f ℥ ii	60.0
	M.		

S.—One teaspoonful every two to four hours, for a child three years old.

Where the cough is very disturbing, it is advisable to add from 1/24 grain to 1/16 grain of codeine to each teaspoonful of the medicine.

Occasionally I find it necessary to alternate this mixture with the following:

℞	Creosoti Carbonatis	℥ ss	2.0
	Glycerini	℥ iv	15.0
	Pulv. et Mucilago Acaciæ q. s.		
	Aquæ Anisi	q. s. ad f ℥ ii	60.0
	M.		

S.—One teaspoonful every three to six hours for a child three years old.

Bronchitis Chronica

Chronic bronchitis is not very common in children. It may occur as a sequel of acute bronchitis or pneumonia, diphtheria, pertussis and heart and kidney diseases. It may gradually give rise to dilatation of the bronchi (bronchiectasis), emphysema or asthma, in which event the symptomatology resembles that of the other affections. A Roentgenogram is often helpful in the diagnosis. This procedure is especially valuable in the detection of foreign bodies in the bronchi and tuberculous foci.

Treatment.—Attention to the nasopharynx and larynx. Inhalation of medicated vapors. Small doses of ammonium iodide or the syrup of hydriodic acid. Change of air. General tonics. (See also Asthma, Bronchiectasis, and Emphysema.)

Broncho or Lobular Pneumonia

Next to gastrointestinal diseases, bronchopneumonia is the most common affection of early childhood. In the majority of cases it is caused by a mixed bacterial infection—of the pneumococcus, streptococcus, staphylococcus aureus, *B*. influenzæ and *B*. tuberculosis. It frequently occurs also secondarily to the exanthematous diseases, pertussis, erysipelas and chronic heart, kidney, and intestinal maladies. Recurrent colds, rachitis and other wasting diseases serve as active predisposing causes.

The onset of bronchopneumonia may be sudden or gradual in association with tracheobronchitis or capillary bronchitis, as a result of extension of the inflammation. The pathologic process is usually bilateral. Small areas of pulmonary congestion, consolidation and resolution are scattered throughout the entire lung. On section the affected lobules present quite a smooth surface of bluish-red color. The bronchioles and pulmonary alveoli are filled with a mucosanguinolent and mucopurulent exudation. The bronchial walls are thickened and infiltrated with small round cells, and the lymph nodes are enlarged and congested. Quite often the pleura is implicated in the inflammatory process.

As already stated, transition of the inflammation from the large bronchi to the fine bronchioles (bronchiolitis) and lung tissue (pneumonitis) not rarely proceeds insidiously, in fact, the bronchopneumonia may exist for a few days before being detected. This holds true especially of bronchopneumonia accompanying influenza, measles and diphtheria. In the majority of cases, however, the onset is ushered in with rise of temperature (up to 105° F.), fretfulness, vomiting, and occasionally convulsions. The cough is dry, short and painful, the pulse and respiration are greatly increased in frequency. A pulse of 130 to 160 beats per minute and a respiratory rate of from 40 to 60 are quite common. There is moderate dyspnea; the alæ nasi are contracting and dilating forcibly; the eyes are dull, and the face is pale and slightly cyanotic. In virulent cases the dyspnea gradually increases, the heart's action becomes weaker, and the patient rapidly succumbs to cardiac exhaustion and toxemia, usually preceded by attacks of tachycardia and tachypnea, coma and convulsions. Even in favorable cases, the course of the disease is usually protracted, lasting from two to four weeks or longer, principally because of repeated extension of the pneumonic process to new areas, not rarely with resolution of the old foci. Furthermore, the course of the disease is often aggravated by numerous complications: as, for example, pleuritis, otitis, stomatitis and gastroenteritis, and quite frequently also by pyothorax. Where resolution is long delayed, bronchopneumonia may also terminate in tuberculosis and pulmonary gangrene.

The physical signs are indefinite in the early stage of the affection. The face is flushed on one or both sides and with each inspiration there is more or less marked retraction of the soft structures in the intercostal and suprasternal spaces. The respiratory sounds are rough and accentuated, and here and there intensified by diffuse small and large sonorous râles. As the disease advances and the localized pneumonic foci multiply, become consolidated and coalesce, we are soon able to detect the typical signs of pneumonia, i. e., dulness on percussion, bronchial breathing, bronchophony and occasional fine crepitation. An undue degree of flatness on percussion should be looked upon as a suspicious sign of pleuritis with effusion.

The prognosis of bronchopneumonia is always very grave, especially in infants under one year of age, in whom the mortality ranges between 20 and 30 per cent. Grave, often fatal, are usually the cases presenting the following symptoms: continued hyperpyrexia, pallor and cyanosis, marked tympanites, dyspnea with respirations irregular in depth and rhythm, coma and convulsions, and recurrent recrudescence of the pneu-

monic process after apparent defervescence. (See also Influenza- Pneumonia.)

Treatment.—Bronchopneumonia being most frequently the sequel of some other serious affection, it is therefore obvious that prophylaxis forms the *sine qua non* in our therapeusis. By viewing every simple nasopharyngeal and bronchial catarrh as a precursor of lobular pneumonia, and by applying the proper means to arrest it at its inception, a great many cases could readily be prevented. Bronchopneumonia, once established, we have no specific to combat it. However, an attempt can yet to be made to modify the virulence of the disease by means of the following procedures. The patient is given a hot mustard bath of about three minutes' duration, is wrapped in a warm blanket, surrounded by a few hot water bags and given hot drinks, moderate doses of sweet spirits of niter or spirit Mindererus, etc., to stimulate free diaphoresis. This is soon followed by the application to the chest and back of a hot poultice consisting of six tablespoonfuls of flaxseed meal, three tablespoonfuls of camphorated oil, one or two tablespoonfuls of powdered mustard and a sufficient quantity of hot water to make a thick paste by thorough stirring. The mass is spread thickly on thin gauze. The child is then wrapped in an oiled silk jacket lined with absorbent cotton and blanket, which with the hyperpyrexia of the body, maintain the heat of the poultice, so that its renewal is required but three or four times in twenty-four hours. The poultice is very useful, especially where the breathing is painful and difficult. In these cases some benefit may be derived from the application of from twelve to twenty-four dry cups. Where the temperature is very high and the poultice is apt to interfere with the hydrotherapeutic procedures, we may resort to mustard cloths (wrung out of a mustard solution, one teaspoonful of mustard to a pint of warm water). The temperature should preferably be reduced by cool sponging, cool pack, ice cap to the head, and where cerebral symptoms prevail, by warm baths, with or without mustard, although an occasional dose of pyramidon, aspirin or phenacetin will do no harm.

The maintenance of the child's strength is most essential to the successful management of the disease. Be it remembered that death in pneumonia is due to heart failure and not to pulmonary insufficiency; therefore, the heart must receive early and diligent attention. We may begin with the tinctures of digitalis and strophanthus (one drop of each for every year of the child's age up to about six years) every four to six hours, and more frequently if the circulatory and respiratory difficulty increases. In bad cases the stimuluation may be intensified by the addition of sterile camphorated oil (3 grains) and strychnine sulphate

(1/60 grain) hypodermically every four hours; and where signs of pulmonary edema supervene, by an occasional dose of atropine sulphate (1/200 grain). In sudden collapse, suprarenal solution (5 to 10 minims hypodermically) is worth trying.

Every effort should be made to replenish the body fluids consumed during the active febrile process by suitable liquid nourishment, such as broths, beef tea, small quantities of milk or fermented milk, fruit juice, etc., in addition to large quantities of water. In extreme cases, saline entero- or hypodermoclysis, and, in older children, saline intravenous may have to be resorted to. The urine should be watched for acetone and pus, the latter especially in girls. Excessive tympanites often yields to intestinal irrigations with bicarbonate of soda solutions (½ ounce to 2 quarts of water) with or without the addition of essence of peppermint (10 to 15 minims) or to pituitary solution hypodermically. This may be repeated two or three times in twenty-four hours. Complications arising should receive prompt attention.

When called upon to treat bronchopneumonia with delayed resolution, our efforts should be directed mainly towards the prevention of empyema or tuberculous infiltration of the lungs. A great deal can be accomplished by placing the patient in a large airy room during the febrile stage, and, weather permitting, keeping him outdoors most of the time, after the temperature has dropped to normal or to a degree above. During convalescence removal to the country is highly to be recommended.

The iodides will often be found very useful to hasten resolution. We usually begin the administration of the sodium or ammonium iodide, in ½ to 2 grain doses, about the sixth day of the disease, and continue it until resolution has been established. After the temperature has disappeared, we give the syrup of the iodide of iron with the compound syrup of hypophosphites, which acts both as an alterative and tonic.

Creosote is indicated in all stages of bronchopneumonia (see Prescription, p. 316). The ordinary beechwood creosote may also be used for inhalation by means of a croup kettle (10 to 20 minims in a pint of hot water). Its effect is intensified if a tent is improvised around the child's bed.

If notwithstanding the aforementioned therapeutic measures the bronchopneumonia fails to resolve, and the physical signs and exploratory puncture fail to disclose pus in the thorax, we must direct our attention to the possible presence of a latent or florid *tuberculous process*. The diagnosis between simple bronchopneumonia due to mixed infection and acute or subacute tuberculous bronchopneumonia is often very difficult. In the tuberculous variety the onset is usually more gradual, the temperature curve more intermittent, the loss in weight more rapid and

the areas of consolidation more stationary in character, and giving rise to more definite physical signs, such as flatness, bronchial breathing, bronchophony, etc. An exact roentgenogram is often decisive in the diagnosis, revealing, as it frequently does, marked involvement of the bronchial glands. The von Pirquet test is usually negative in the non-tuberculous form.

Lobar Pneumonia

(CROUPOUS PNEUMONIA, FIBRINOUS PNEUMONIA, PNEUMONITIS, PNEUMOCOCCUS PNEUMONIA)

Acute lobar pneumonia is a primary, specific, communicable, occasionally epidemic, affection of the lungs, pathologically characterized by pulmonary engorgement, red hepatization, gray hepatization and resolution. The pneumococcus or diplococcus lanceolatus of Fränkel-Weichselbaum, the immediate cause of lobar pneumonia, can readily be isolated—usually in pure culture—in the sputum, lung substance, and the blood, in four different groups—Type I, II, III, IV.*

Pathology.—In the stage of *engorgement* or congestion the lungs show very little that is characteristic. They are dark red, still contain air, but are slightly firmer in consistence, and resemble mostly a beginning hypostatic pneumonia. Without the aid of the microscope it can be anatomically diagnosed best when fibrinous hepatization is to be seen immediately adjoining it.

In the stage of *red hepatization* the alveoli become filled with red blood corpuscles and fibrin. On coagulation of the fibrin the hemorrhagic contents of the alveolus become a quite firm, red plug. The cut surface of red hepatization is red and slightly granular. The latter gradually changes to grayish-red and, in part, grayish-yellow (gray hepatization). This is due to solution of the blood corpuscles, diffusion of the blood coloring matter, and exudation of new fibrin masses and partly also of cellular elements into the alveoli. The hepatized area thus attains a volume as in deep inspiration, with the difference, however, that instead of air a firm exudate occupies the alveoli which produces anemia of the lung tissues as a result of pressure upon the vessels. If the edge of a knife is held at a slant and scraped across the cut surface, grayish-yellow granules are obtained, which are composed of a dense network of fibrin inclosing a moderate number of colorless blood corpuscles and a few desquamated alveolar epithelia. This is the stage of complete hepatization.

---

*Blanke and Cecil (Jour. Exp. Med., April, 1920) have shown that lobar pneumonia is bronchiogenic in character. Invasion of the blood stream by pneumococci is secondary.

The cut surface gradually becomes smoother and redder, and the solid consistency gives place to a more relaxed condition. If the cut surface now be scraped with the edge of a knife, a cloudy fluid, partly mixed with solid masses, is seen, which consists microscopically of finely granular detritus, disintegrated cells and a few large, still coherent clumps or plugs. These plugs contain chiefly round cells and only a slight amount of fibrin. This is the stage of *resolution, i. e.,* loosening, softening, transformation from the solid to the fluid state; the exudation is partly expectorated and partly absorbed, as a result of a fermentative, proteolytic process (R. Langerhans and H. T. Brooks).

Lobar pneumonia is generally accompanied by fibrinous pleuritis, and more or less marked bronchitis. As a rule, only one pulmonary lobe is affected, and the lower right more frequently than the others. If several lobes are involved, it usually occurs by successive invasion.

Primary fibrinous pneumonia usually ushers in suddenly, often after exposure to cold or wet, with vomiting, chilliness, high temperature and more or less marked dyspnea. The initial symptoms are frequently misleading. They may consist of vomiting, diarrhea, pain in the abdomen and nosebleed, suggesting the beginning of typhoid fever; or convulsions, sopor, vomiting and severe muscular pain may predominate, justifying the tentative diagnosis of meningitis. Where the pneumonic lesion is located centrally (so-called central pneumonia), and the physical signs, nay, even the cough, is absent or very slight in the early stages of the disease, one is not rarely tempted to diagnose remittent malarial fever. Furthermore, there are also numerous cases of pneumonia of only a few days' duration (so-called abortive pneumonia), which undoubtedly escape observation or are recognized only by their critical defervescence.

Of course, the majority of cases pursue a typical course and are readily elicited on careful physical examination. As a rule, auscultation discloses harsh breathing all over the chest, and during the first stage often distant breathing over the affected area and fine crepitation along its edges. In the second stage, when the consolidation is complete, the breathing is distinctly tubular and the vocal resonance bronchial in character (bronchophony). In the third stage, with beginning resolution, fine crepitant râles (crepitatio redux) return, but are often softer in quality. Bronchophony may continue long into convalescence. In the first day or two of the disease the percussion sound is usually tympanitic—owing to the presence of some air in the involved lung, but as the consolidation advances, we readily elicit dulness or flatness, the experienced hand perceiving also a distinct increased sense of softness and resistance which is transmitted to the
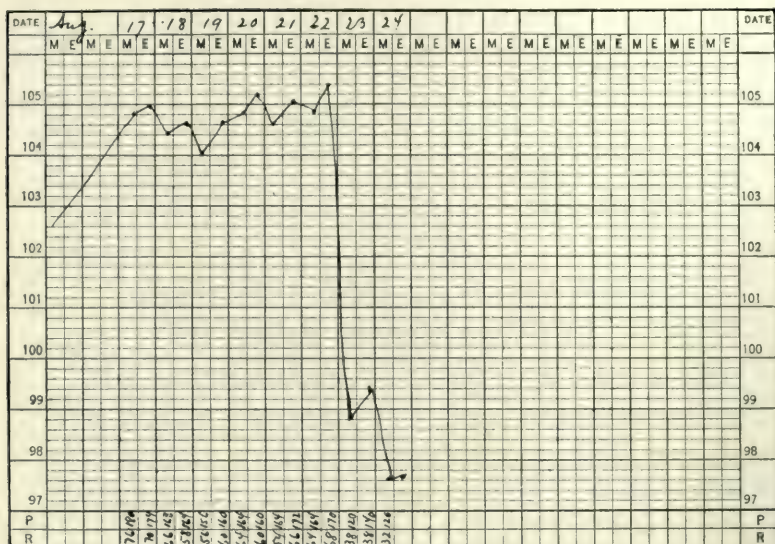
Fig. 73.—Fever curve of typical lobar pneumonia in a child fourteen months old, ending by crisis.
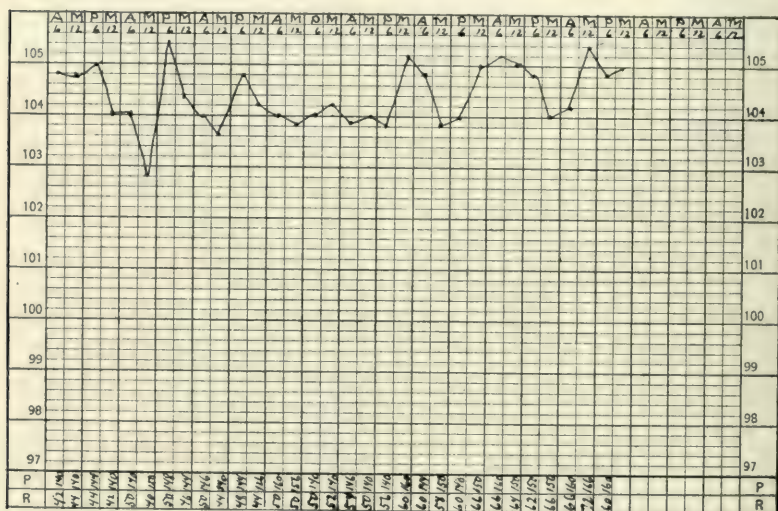

Fig. 74.—Fever curve of a fatal case of apex pneumonia with marked cerebral symptoms in a child two years old.

percussed finger. Pectoral fremitus is ordinarily not pronounced in young children, except when they cry aloud, which act should always be encouraged to facilitate the exposition of the physical signs. In all stages of pneumonia, inspection reveals more or less marked dysp-

nea, dilatation of the alæ nasi, and depression of the peripneumonic groove with each inspiration.

Croupous pneumonia generally runs a self-limited course, from five to thirteen days or longer, and most frequently terminates by crisis, at a time when the disease is at its height. Until then, especially in the absence of complications, there is little change in the clinical picture of the affection. The fever remains high (104° to 105° F. or higher) with slight remissions; the pulse and respiration ratio is greatly disturbed, from $1:2\frac{1}{2}$ to $1:2$; the cough short, dry and painful, and in older children often attended with rusty expectoration; the face is pale with a hectic flush and the nose and lips are more or less cyanotic; the urine is scanty, highly colored, rich in salts (with diminution in chlorides) and occasionally in peptone and acetone; the tongue heavily coated, sometimes blackish with its tip red; and finally the abdomen is considerably distended. The child is very restless, listless, tosses from side to side, and when able to respond to questions, usually indicates the seat of pain somewhere in the abdomen, usually on the side where the lung is affected.

As the crisis approaches and the circulatory difficulties become more and more pronounced, the expiratory moan becomes louder and longer, the cough more harassing, the breathing more superficial, the pulse more rapid and feeble, the thirst more intense, and the sensorium more disturbed,—the child lies helpless, often rigid, in a state of apathy, frequently interrupted by incoherent outcries and on the verge of collapse. The change wrought by the establishment of the crisis is certainly miraculous.* In but a few minutes the heart's action calms down (the pulse is often irregular and very feeble), the breathing becomes slower and deeper, and the patient bathed in perspiration falls into a more or less profound sleep, from which he often awakens fully refreshed, free from pain and fever (sometimes 2 or 3 degrees below normal), ready to take nourishment, and happy to start life anew.

Unfortunately the typical course of the disease is often marred by a number of complications, and even without these, pneumonia may prove fatal, the mortality ranging between from 10 to 20 per cent. Not rarely, lobar pneumonia terminates by lysis. Defervescence may be tardy, the temperature reaching normal by two or three stages. Occasionally, after an apparently true crisis and durable defervescence, recrudescence takes place, the temperature again rising and continuing for several days. In some instances where the pneumonia is greatly

---

*The crisis and resolution of the pneumonic process seems to be due to local biochemic changes, in the course of which, as suggested by Lord (Jour. Exp. Med., October, 1919), the acid death point of the pneumococcus is reached.

protracted, the inflammatory process is found to "creep" from lobe to lobe, and may end either in gradual recovery or in unresolved, chronic or the so-called caseous pneumonia. Very serious also are the cases in which the lung involvement is very extensive (double pneumonia) from the start; where the inflammation, spreading to the pleura (pleuropneumonia), gives rise to free effusion which, through secondary infection, terminates in pyothorax; and where the pneumonia supervenes upon other infectious diseases (e. g., influenza, diphtheria), more especially if it is preceded by cardiac exhaustion from the effects of the underlying affection. Occasionally fulminating pneumonia is met with, which is manifested by extreme dyspnea, cyanosis, tympanites, high continuous temperature, very rapid and feeble pulse, and cerebral symptoms, and ends fatally within two or three days. These cases are usually due to mixed infection.

Complications also substantially mar the prognosis as regards the immediate and ultimate recovery, meningitis and pericarditis proving particularly disastrous. As already stated, pleurisy, with a serous and more especially purulent effusion, is more apt to influence the prognosis at a later stage. Pneumococcus peritonitis is a very grave complication, but recovery may ultimately ensue if the pus in the peritoneal cavity becomes encapsulated and finds its way out either through the intestines or the umbilicus. Suppurative foci (pneumococcic metastases) are occasionally encountered in the bones and joints and ordinarily yield to surgical procedures. Among other complications, we may also mention otitis media, which usually clears up with or without perforation.

The relation between a high leucocytosis and favorable prognosis in lobar pneumonia is still subject to controversy. Generally fibrinous pneumonia is associated with a high leucocytosis, the proportion of the white cells to the red ranging anywhere between 1 to 40 to 1 to 70, which is nearly twice as high as in the lobular variety. According to Koplik, a very low leucocyte count with marked signs of pneumonia and high temperature is a grave prognostic sign. Such cases, however, may recover. On the other hand, even a high leucocytosis, with extensive inflammation of both lungs, does not prevent a fatal issue.

Differential Diagnosis.—In the initial stage lobar pneumonia may be confounded with bronchopneumonia, pleurisy, meningitis, and appendicitis; in the second stage with pleurisy with effusion, and in the absence of cough and the presence of marked tympanites, with peritonitis; and in the third stage, especially where resolution is delayed, with miliary tuberculosis. Moreover, concurrence of pericarditis may occasionally obscure the original disease. Careful attention to the pathognomonic signs and symptoms of the different affections ought to

readily clear up the diagnosis. We must, however, always bear in mind the fact that any of the aforementioned diseases may at any time complicate the pneumonia. In doubtful cases, a careful differential count, and, in hospital practice, an x-ray examination will often facilitate the diagnosis.

Acute Lobar Pneumonia	Catarrhal Pneumonia
Generally a primary disease.	Secondary.
Onset sudden.	More gradual.
High regular fever.	Moderate and irregular.
Inflammatory process localized.	More diffuse.
Physical signs distinct.	Indistinct.
Termination by crisis, the rule.	By lysis.

Acute Lobar Pneumonia	Miliary Tuberculosis
Onset sudden and marked.	More gradual and masked.
Fever high and regular.	Very irregular.
Tuberculin test negative.	Positive, as a rule.
Complement-fixation test negative.	Positive in early stage.
Sputum contains pneumococci.	Tubercle bacilli.
Duration from one to two weeks with tendency to recovery.	From three to six weeks, ending fatally.
See also Pleurisy, page 328.	

Treatment.—Pneumonia being a communicable affection, it calls for all such hygienic precautions as are ordinarily employed in the prevention of other contagious and infectious diseases. (See p. 68.) The sputum should be collected in small pieces of gauze and destroyed.

Fresh cool air is the *sine qua non* in the management of pneumonia. It purifies the respiratory tract, eases respiration, facilitates the pulmonary circulation, hence relieves and regulates the heart's action, reduces temperature, and cheers the patient in those endless, wakeful hours, which are so characteristic in pneumonia, and last but not least, disinfects the sick room and thus prevents transmission of the disease to others, as well as autoinfection of the patient.

Plenty of pure drinking water is the next most important requisite. It should be given *ad libitum,* unless contraindicated by uncontrollable vomiting. Pure water cleanses the mouth, pharynx and alimentary canal which in children with pneumonia are usually infected by the large quantities of putrid sputum that are swallowed rather than expectorated; it quenches the ever-present agonizing thirst; stimulates expectoration and aids in reduction of temperature.

It is advantageous to administer daily a low enema containing a quart or two of warm water with an ounce of bicarbonate of soda—to cleanse the bowels and to relieve intestinal fermentation, and also to counteract the acetonuria which is quite common in febrile affections. Saline re-

tention enemas (105° F.) may also be given a few times daily to stimulate the action of the kidneys and heart.

Water should serve as the only antipyretic when reduction of fever is indicated, *i. e.*, if above 102° F., and may be administered in the form of cold sponges, cold packs, warm baths and, in older children, even cool baths followed by brisk friction, if the temperature remains persistently high. In excessive nerve irritability mustard may be added to the warm bath (see "Hydrotherapy," p. 90), although in such cases an occasional dose of sodium bromide (5 grains) and pyramidon (2 grains) will often act very beneficially.

Pain and cough in pneumonia may be readily relieved by minute doses of codeine, with or without sodium salicylate, or by local heat, either in the form of a flaxseed and mustard poultice (see p. 318), or cloths immersed in warm mustard water, wrung out and covered with oiled silk and towel. The mustard cloths may be changed every half hour until the pain is relieved. In some cases I observed very good results from dry cupping.

Some authors claim that quinine exerts a specific action in pneumonia. It must be pushed to its full physiologic effect—1 to 2 grains for every year of the child's age, every two to four hours. In severe cases, quinine urea hydrochloride may be administered intramuscularly.

The heart action requires careful watching. Where the pulse is very rapid, the tinctures of digitalis and strophanthus (one drop for every year of the child's age) may be given alternately every three hours, while when the pulse drops below one hundred per minute, strychnine sulphate (1/200 to 1/100 grain hypodermically) is the remedy of choice. The "Murphy drip" is a valuable stimulant to the heart and kidneys. For quick stimulation caffeine sodium benzoate (1 to 3 grains every six hours) should also be resorted to. The profession is generally in favor of sterile camphorated oil (3 grains), although I believe that its therapeutic effect is greatly exaggerated. In threatening pulmonary edema, nitroglycerine (1/200 grain) may prove efficient. It is well to bear in mind, however, that undue overcrowding of stimulants may do harm. In cyanosis, oxygen is beneficial. In severe cases of tympanites, pituitary solution hypodermically is well worth trying.

Protracted and unresolved pneumonias often respond promptly to the administration of ammonium iodide (1 grain three times a day for every year of the child's age).

The diet should be light and easily digestible, consisting mainly of broths, fruit juices, small quantities of plain or fermented milk, albumin water, well diluted wine or cognac. Milk should be dispensed with where tympanites is marked.

The mouth and nasopharynx should be gently cleansed several times daily.

The profession is still at odds over the usefulness of vaccines and serums in the treatment of pneumonia. If the type of the pneumococcus can readily be determined and the homologous serum obtained, it could safely be administered, even though we have as yet no positive evidence of its specific effect.

In slow convalescence it is advisable to send the patient to the mountains or to a mild seashore resort and to administer creosote by mouth as well as by inhalation. The syrup iodide of iron and cod liver oil are indicated especially in young children. See "Bronchopneumonia".

Pleuritis

(Pleurisy. Empyema)

The pleura, like other serous membranes, may be affected, *primarily* as a result of trauma, or invasion of pathogenic bacteria, such as the pneumococcus, streptococcus, the microbe of rheumatism, influenza, etc., or *secondarily* by extension of an inflammation from neighboring structures. Primary pleurisy is comparatively rare in young children. The secondary variety, however, is quite common in connection with pneumonia, influenza, tuberculosis, acute heart disease, general sepsis, and affections of the abdominal organs.

Pathologically, pleuritis is characterized by congestion and roughness of either the parietal or visceral layer of the pleura or of both; a fibrinous exudation upon the pleura; in severe cases, a more or less large collection of (serous, serosanguinolent, or seropurulent) fluid between the surfaces of the pleura, or between the gaps and meshes of the fibrinous exudation. In accordance with the extent and location of the pleural effusion, there is more or less severe displacement of the contiguous structures.

I. Dry Pleurisy

It is quite probable that many cases of dry pleurisy in young children escape detection. This is apt to occur especially in secondary pleurisy, where the symptoms of the original disease obscure those of the complication. Moreover, little patients often refer the pathognomonic "stitch pain" to the abdomen instead of to the side. Apart from the pain, the subjective symptoms are few and mild. The child instinctively abstains from coughing and deep breathing, and like an adult lies on the affected side. As a rule, the diagnosis can readily be made on hearing the pleuritic friction sound—a dry, crackling sound

on inspiration. The termination of dry pleurisy is either in rapid
and uneventful recovery (sometimes leaving behind slight pleural thick-
ening and adhesions) or in the graver form of the malady, *i. e.*, in
pleurisy with effusion.

II. Pleurisy With Effusion

A perceptible pleural effusion, be it composed of serum, blood and
serum, pus or chyle may generally be recognized by the following
distinctive features:

Inspection.—Dyspnea with impairment of movement of the affected
side.

In large effusions, fullness of the intercostal spaces and later bulg-
ing of the affected area of the thoracic wall, and not rarely promi-
nence of the hypochondrium of the corresponding side. Occasionally,
enlargement of the subcutaneous veins, and superficial edema. In
cases of long standing in which effusion undergoes partial or com-
plete absorption (as well as after operative removal of the fluid), there
is a lateral curvature of the spine, incurvation of the affected side with
compensatory bulging of the unaffected side of the chest.

Palpation.—As compared with the healthy side, there is distention
of the intercostal spaces on inspiration and diminution of vocal frem-
itus. In large serous effusion, fluctuation may be perceived by placing
one finger of one hand in the intercostal space, and with the finger
of the other hand imparting quick but gentle impulses to the fluid, in
the direction of the other finger.

Auscultation.—Varying with the amount of pleuritic effusion or
thickening, the respiratory sounds may be diminished or absent over
the affected side and exaggerated over the healthy portions of the
lung. Where the effusion is small and the larger bronchi remain open
for the respiratory current of air, we may hear distant bronchial
breathing. In rare cases, especially in tuberculous pleuritic effusion,
the respiratory murmur may simulate cavernous breathing and lead
to errors in diagnosis, especially if the bronchophony over the com-
pressed lung is transmitted along the pleuritic adhesions or the chest
wall.

Percussion.—Dulness or flatness, corresponding to the amount of
pleuritic thickening or effusion, over the affected portion of the lung,
and often tympanitic resonance over the retracted lung tissue. Per-
cussion must be performed lightly; for in the presence of only a thin
layer of fluid, forced percussion may elicit the normal resonance of
the underlying lung. The sense of resistance to the finger is greatly
increased. Displacement of the neighboring organs.

Grocco's sign (paravertebral triangle of dulness on the side opposite to that of the effusion) is rarely elicited in young children, but is of diagnostic value if found.

Roentgen-Ray Examination.—In the majority of instances an x-ray examination aids greatly in localizing the fluid in the thoracic cavity, especially when the effusion is scanty and encapsulated or is located under the diaphragm (subphrenic abscess). In this connection it is

Fig. 75.—Grocco's sign of pleurisy with effusion (paravertebral triangle of dulness on the side opposite to that of the effusion—*G*).

important not to interpret the shadow of gas in the upper gastro-intestinal tract as fluid in the thoracic cavity.

With the establishment of the presence of a pleuritic effusion by means of the aforementioned physical signs, the nature of the pleural fluid content still remains to be determined. In the majority of instances this can readily be accomplished by means of exploratory puncture.

Except where the exudate is buried behind a thick pleural mem-

brane or, more rarely, behind tumors of the chest wall (so that the needle does not reach the fluid), or where the pleural content is too thick to pass through the needle, exploratory puncture of a pleural effusion usually reveals any of the following fluids: serum, serum with blood, serum with pus, pure pus, or chyle. In accordance with these findings, it is customary to distinguish serous or serofibrinous pleurisy, hemorrhagic pleurisy, purulent pleurisy (empyema, pyothorax), and chylothorax.

Serous or Serofibrinous Pleurisy

The onset may be sudden with vomiting, chills, rise of temperature and pain in the side, or, more frequently, insidious,—either as a primary disease with general malaise, short cough, increasing dyspnea and pallor, or as a secondary affection, with accentuation of the symptoms of the primary disease. In acute pleurisies the fever may be moderately high and persist for from two to three weeks, and then gradually subside, even though the effusion remains. Bilateral pleurisy is almost always tuberculous. Pleurisy, associated with pericardial or peritoneal symptoms, points to its tuberculous character. In young children with a yielding thorax, absorption of large effusions is, as stated, almost always associated with contraction of the affected half of the chest. The ribs become pressed together, the intercostal spaces narrowed, the shoulder blade is drawn nearer the vertebral column, and the latter is curved (scoliosis). With complete recovery from the disease, the deformity may in some cases gradually disappear. In the majority of instances, dulness and suppressed respiratory murmur continue as a result of pleuritic thickening.

The prognosis of this form of pleurisy, except that due to tuberculosis, is generally favorable. Occasionally acute pleurisy terminates fatally either as a result of a sudden excessive effusion, or of pulmonary edema, embolism of the pulmonalis or of a cerebral vessel.

Hemorrhagic and Tuberculous Pleurisies

In the recent epidemic of influenza quite a number of children presented a hemorrhagic exudation in the pleural cavity, in connection with bronchopneumonia. In one case, a boy nine years old, we aspirated over two quarts of hemorrhagic fluid which showed the streptococcus hemolyticus in pure culture. The patient succumbed to the disease within a week notwithstanding early thoracotomy and the administration of autogenous vaccine.

Protracted cases of pleurisy should always be looked upon with suspicion. In very many instances they are of tuberculous nature. This

is particularly true of bilateral pleurisy and of that with prolonged irregular temperature and serohemorrhagic exudation. It is well to remember, however, that a hemorrhagic effusion is sometimes observed in scorbutic children, and that puncture of a blood vessel or injury to the diaphragm or liver may bring forth blood in the aspirating syringe. In tuberculous pleurisy, before long, other symptoms of tuberculosis make their appearance. The presence of the tubercle bacillus in the exudate, or, if the lungs are involved, in the sputum, and a positive tuberculin test settle the diagnosis.

Purulent Pleurisy (Empyema, Pyothorax)

Owing to the frequency of pneumonias (the principal cause of pleuritic effusions) in children, empyema is of very common occurrence. In the majority of instances the exudation is purulent from the beginning, more rarely it is serous at first, and, after a protracted course, undergoes suppurative transformation, as a result of an endogenous infection by the pneumococcus, streptococcus, staphylococcus, or the tubercle bacillus. Pyothorax is usually unilateral, and localized on the left side more frequently than on the right. Occasionally it is bilateral, e. g., in sepsis, pyema, etc. Still more rarely it is multilocular, encysted, or interlobular. The amount of pus varies from a few teaspoonfuls to a quart. The exudate may on the first puncture prove to be seropurulent; but, as the disease advances, the purulent character increases, becoming greenish-yellow in color and sometimes fetid in odor. It may be feculent, indicating some connection with the abdominal contents.

Pyothorax may also develop primarily as a result of trauma. As a rule, however, it is met secondarily to inflammatory, especially suppurative, processes of the thoracic and abdominal organs, of the joints, ribs and vertebræ, or in association with general sepsis. As a sequal or complication of thoracic or abdominal diseases, empyema usually sets in very insidiously, and may even remain latent for some time until either the effusion is so large as to cause bulging of the affected side of the chest, or to be discovered accidentally during a routine examination for some other ailment. The onset is more acute in cases due to trauma, necrosis of the neighboring bony structures, exanthematous diseases, or in sudden rupture into the pleural cavity of abscesses of the neighboring organs (e. g., hepatic, perinephritic, etc.). In such cases the symptoms resemble those of acute serofibrinous pleurisy, except that the temperature is higher and more irregular and emaciation and exhaustion are more pronounced.

Aside from the physical signs already enumerated, empyema complicating pneumonia may generally be suspected where resolution is delayed and the temperature continues high and irregular and is accompanied by sweating. In such cases, even in the absence of pathognomonic physical signs, Roentgen-ray examination and exploratory puncture should not be long delayed.

Fig. 76.—Extensive right empyema in a child four years old.

With early operative treatment empyema in children usually terminates in recovery. If let alone, the abscess may rupture spontaneously either in the lungs or externally through the chest wall—*empyema necessitatis.* The point of external rupture is usually found in the vicinity of the sternum, where the chest wall offers least resistance. If the rupture is into a bronchus, a very large expectoration of

pus occurs suddenly. In these cases there is always danger of pyopneumothorax. In another group of cases the pus may by inspissation lead to caseous residues and to fatal issue from gradual exhaustion or from complications, such as tuberculosis, amyloid degeneration, etc.

Chylous Pleuritis (Chylothorax)

Genuine chylous effusion in the thorax is an exceedingly rare condition. More frequently we meet with other milky effusions,—chyliform, latescent (nonchylous). True chylous effusion is the result of injury or obstruction of the thoracic duct, allowing the escape of chyle either directly through an opening in the wall of the duct or indirectly by transudation.

The *differential diagnosis* between the different varieties of pleurisy can readily be made by means of an exploratory puncture, and by chemic, bacteriologic, and microscopic examinations of the fluid obtained. Bilateral (usually tuberculous) pleurisy may be confounded with hydrothorax. The latter condition, however, is associated with anasarca, consecutive to heart or kidney disease, and generally runs an afebrile course. Left-sided pleurisy may be differentiated from pericarditis by the absence of heart symptoms (triangular heart dulness) in the former, and of lung symptoms in the latter. The synchronous occurrence of both of these diseases, however, should be borne in mind. Right-sided, purulent pleurisy may be mistaken for an abscess or hydatid cyst of the liver. Careful examination will elicit the following differential points: in liver affections the midaxillary line forms the highest point of dulness; there are fluctuation, local tenderness and icterus; in pleurisy with effusion the last-named signs are absent and the midaxillary line forms the lowest point of dulness. Furthermore, in pleurisy aspiration brings forth serum, blood or pus: in hydatid cyst of the liver, a nonalbuminous fluid with "hooklets."

The differentiation between lobar pneumonia and pleurisy is not always easy, since both diseases often coexist. In the latter event, however, exploratory puncture will readily clear up the diagnosis.

PNEUMONIA	PLEURISY WITH EFFUSION
Dulness (late).	Flatness (early).
Temperature high.	Low (in absence of pus).
Pulse-respiration ratio greatly disturbed.	Not so.
Bronchial breathing, bronchophony.	Suppressed breathing.
Vocal fremitus and resonance increased.	Diminished.

Treatment.—During the acute stage, keep the patient in bed. Limit the supply of fluids (in older children a semisolid diet, consisting

principally of cereals, concentrated broths, beef juice, soft-boiled eggs, etc.). Relieve pain by salicylates, perhaps with some opiate internally; by strapping of the chest; flaxseed poultices, or the following ointment

> Tr. Iodini
> Ol. Gaultheriæ
> Ol. Terebinthæ
> Guaiacolis
> Ichthyolis   aa. ℥ i   │   4.0
> Liq. Vaselini   q. s. ad f ℥ ii   │   60.0
> M.
>   S.—Paint the affected parts twice a day, cover with absorbent cotton and bandage.

Should the exudation increase to such an extent as to greatly interfere with breathing, aspirate with Potain's aspirator and follow it up with the local application and strapping, and the administration of sodium iodide and infusion digitalis—the iodide to promote absorption of the fluid, the digitalis to counteract the interference with the heart action by the exudate, as well as to stimulate diuresis. These latter procedures (except aspiration) are indicated also in cases running a protracted course, even without a large effusion. Aspiration should be practised in tuberculous pleurisy only to relieve the respiratory difficulty, and in chylothorax, both as a palliative as well as a curative measure.

As soon as pyothorax is detected, an immediate operation for removal of the pus is imperative. To wait for eventual spontaneous evacuation of the pus through the lungs or externally, is hazardous, principally because of the supervening, often fatal, exhaustion, and of the danger of complicating pyopneumothorax, an incurable fistula, or caseous degeneration. It is to be noted, however, that in acutely developing influenza empyema aspiration is to be preferred to thoracotomy until the active pulmonary inflammation has subsided. In tuberculous empyema, surgical interference is indicated only in threatening suffocation, or grave cardiac embarrassment. Empyema of brief duration with readily flowing pus usually does well with a free incision into one of the intercostal spaces and good drainage. On the other hand, cases of long standing or those with inspissated pus should be treated by resection of a rib, in order to permit free escape of the pus. The disfigurement after such operation in children is comparatively slight, and many cases of regeneration of even several ribs are on record. If the empyema is bilateral, it is advisable to operate at separate sittings.
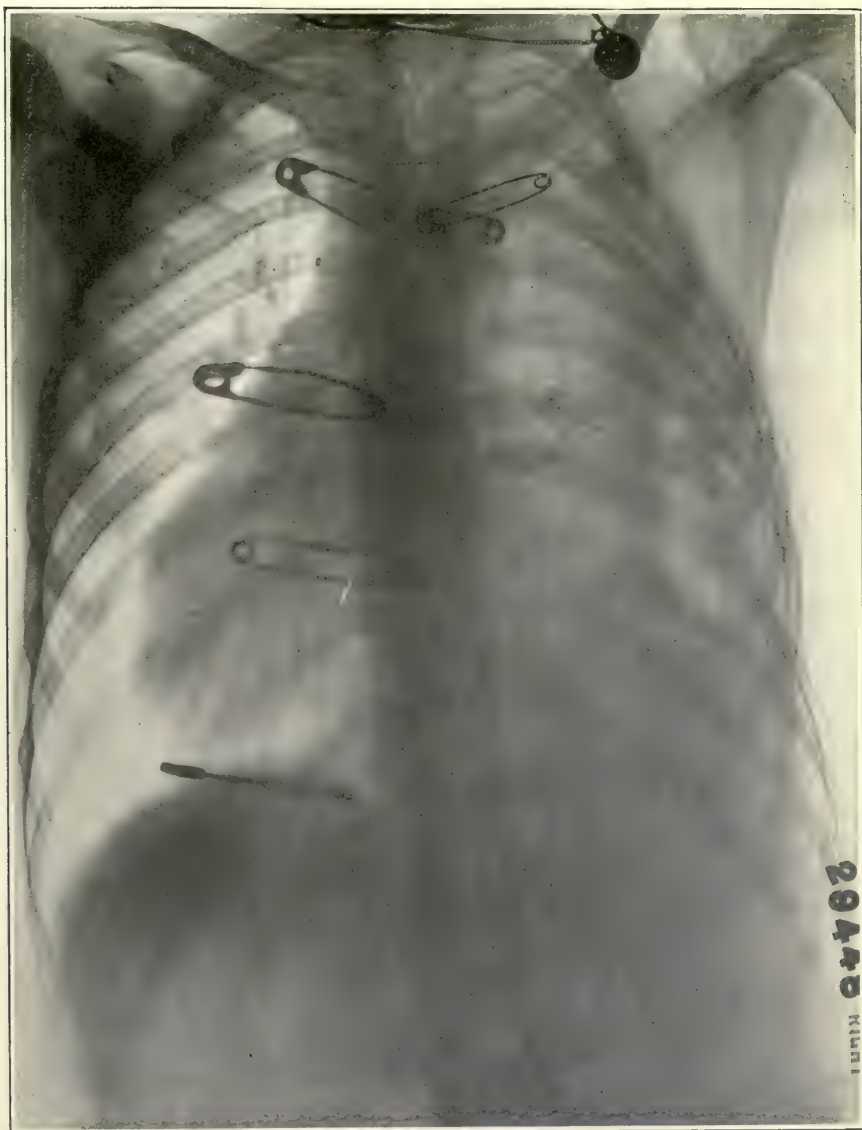
Fig. 77.—Same case as Fig. 76 three weeks later after resection of second and third ribs.   Note clearing of right lung.

Patients recovering from pleurisy, with or without effusion, should have plenty of outdoor air, preferably in the country, seashore. or mountains.   Older children will derive great benefit from horseback riding.   For expansion of the retracted lung after a protracted attack of pleurisy with effusion, systematic breathing exercises and cold sponging of the chest or cold affusions are very useful.
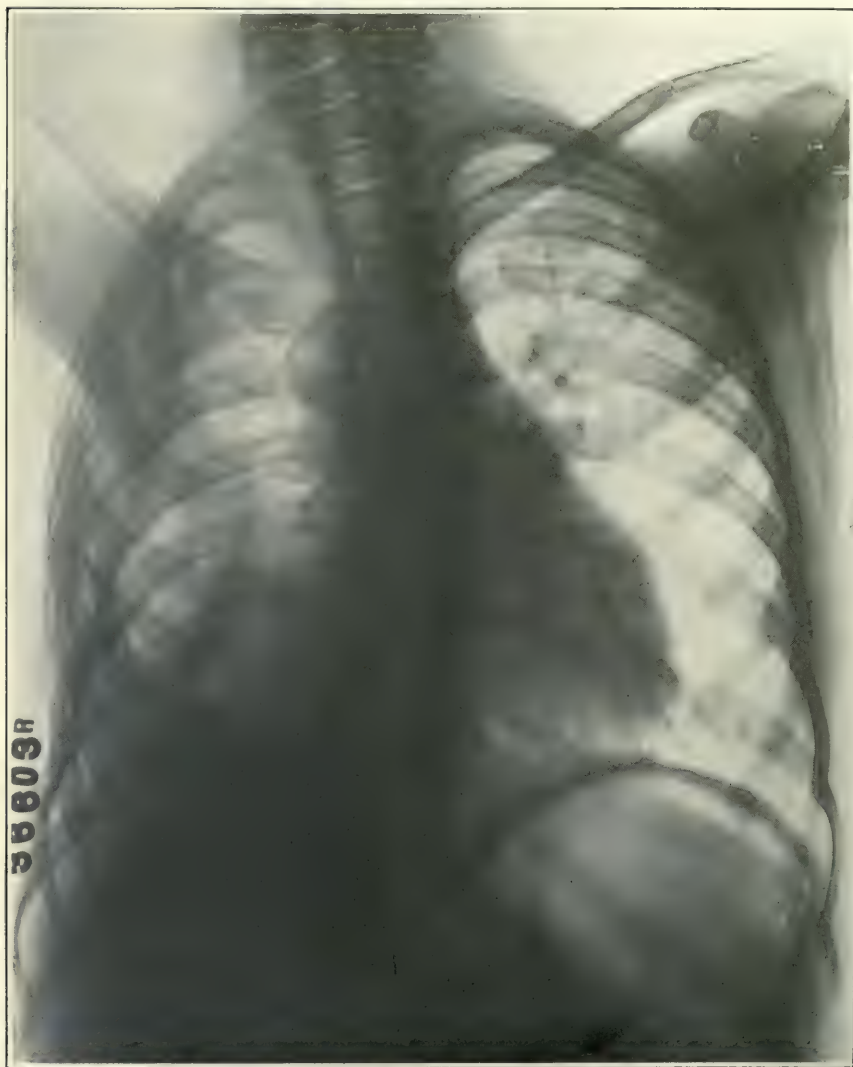
Fig. 78.—Same case as Fig. 76 two months later. Right lung field almost clear.
Note retraction of chest wall and secondary scoliosis.

The importance of wholesome feeding should not be underestimated.
Iron, the hypophosphites, cod liver oil, and extract of malt are helpful
to effect the cure.

Prompt attention to suppurative foci (*e. g.,* necrosis of ribs or ver-
tebrae) and early treatment of pneumonia by fresh air will frequently
prevent empyema.

Asthma

The pathogenesis of asthma in children is essentially the same as that in adults—stenosis of the lumen of the bronchial tubes. The stenosis may be brought about either by a spasmodic contraction of the muscle fibers of the bronchioles, or by vasomotor turgescence and swelling of the bronchial mucosa. Children suffering from asthma usually present an hereditary tendency toward the disease, a susceptibility to protracted irritations of the nasopharyngeal, laryngeal, and bronchial mucous membranes (exudative diathesis, *q. v.*,) or a history of pertussis, bronchopneumonia or chronic bronchitis. In many instances local causes, such as adenoids, deformities of the nasopharynx, persistent thymus, etc., are met with, and some cases are traceable to reflex causes, *e. g.*, indigestion (see "Allergy," p. 87). Asthma in young children seems also to be correlated to eczema. In one marked case (two-year-old baby) under my observation, recurrence of the asthmatic attack coincided regularly with the subsidence or marked improvement of the eczematous eruption. Symptomatic asthma is occasionally based upon *hay fever*—resulting from the action of pollen of certain plants upon the mucous membrane of the nasopharynx—and, finally, an asthmatic attack is sometimes a manifestation of hysteria.

CLASSIFICATION OF CAUSES OF BRONCHIAL ASTHMA.
BRONCHIAL ASTHMA (WALKER)

AGE AT ONSET OF ASTHMA	NUMBER OF CASES	PER CENT OF CASES	NUMBER SENSITIVE	PER CENT SENSITIVE	NO. SENSITIVE TO PROTEIN IN			
					ANIMAL HAIR	FOOD	BACTERIA	POLLEN
Under two years..	34	9.0	28	83	19	23	5	15
Between 2— 5..	30	7.5	27	90	14	9	2	6
" 5—10..	37	9.0	15	40	12	5	2	15
" 10—15..	35	9.0	24	70	6	5	5	13

Sensitive to proteins — Not sensitive to proteins

Asthma throughout the year — Seasonal asthma — Asthma throughout the year — Seasonal asthma

due to — due to — due to — due to

animals
food
bacteria

pollens

bacteria

bacteria
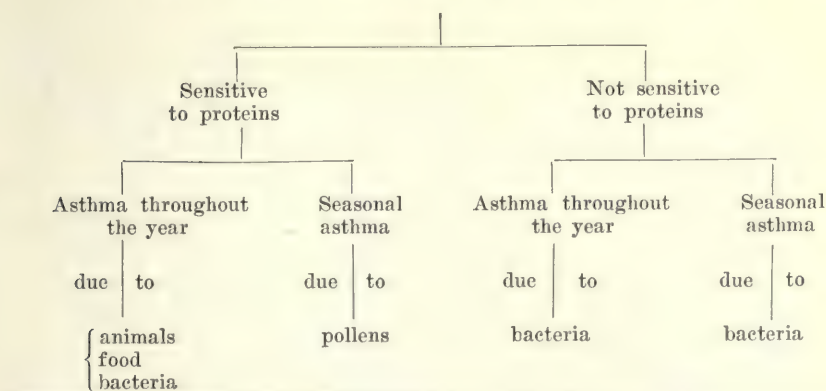
With these etiologic factors in view, the subdivision of asthma into true and false is quite justified. Clinically the two varieties differ in that genuine asthma is invariably associated with chronic bronchial catarrh, hence, is based upon a pathologic entity, and is of longer duration than false asthma. There is nothing characteristic about the catarrh. The paroxysm usually comes on at night. The child coughs, is a little wheezy, and in a few hours the typical attack is in full sway. The latter consists of extreme dyspnea, inspiratory as well as expiratory, anxious expression of the face, congested eyes, cyanosis or pallor, cold extremities, restlessness and prostration. The patient is usually relieved by sitting up in bed. Auscultation of the chest reveals sonorous and sibilant râles, wheezing, squeaking, and whistling respiration. These sounds are often audible at a distance. As the attack subsides the breathing becomes less and less noisy, less labored, and less rapid.

There may be complete apyrexia, or a rise of temperature of from two to three degrees. The respiratory rate may be anywhere from 40 to 80 and the pulse 150 or over. During the height of the paroxysm there is marked eosinophilia, and where expectoration is abundant Curschman's spirals and Charcot-Leyden's crystals are found in the more or less glairy mucus. Toward the end of the attack the thorax may appear barrel-shaped; but unless the asthma is chronic in nature and characterized by prolonged attacks, the emphysematous deformity of the chest is usually only temporary. The attack may last minutes, hours, or days, with temporary remissions, but after abatement of the paroxysm the child is apparently in good health except for the bronchial catarrh. In genuine asthma, exacerbations usually occur in the fall and spring, when the sudden atmospheric changes contribute to catarrh of the mucous membrane of the respiratory tract. On the other hand, paroxysms of false, spasmodic asthma may occur at any time when the exciting cause, e. g., indigestion, sudden fright, etc., presents itself.

As a rule, asthma is not fatal *per se*. Delicate infants, however, may succumb during a severe attack, as a result of suffocation, or after frequently repeated attacks, as a result of emphysema, cardiac dilatation, or even cerebral hemorrhage.

Treatment.—The importance of curing the disease at its very inception or, at least, preventing or mitigating the paroxysm, is obvious. A cure can be effected, if the cause can be found and corrected. Attention to abnormalities of the nose and throat is especially fruitful in this direction. Children having an asthmatic or arthritic history should be given particular care in the way of preventing colds and coughs, overfeeding, exposure to unhealthy surroundings, miasmatic

affections, undue excitement, etc. An attack may, in a way, be aborted by early administration, preferably hypodermically, of atropine, 1/2000 grain and morphine 1/60 grain or adrenalin (5 to 10 minims), and by apomorphine 1/50 to 1/100 grain, repeated, if necessary, after half an hour. The latter drug is especially efficient in "dyspeptic" or "hysterical" asthma. A few drops of a suprarenal gland solution instilled several times a day into the nose sometimes act admirably. If the paroxysm continues we may resort to the following combination:

℞	Potassii Bromidi	ʒ ss	2.00
	Tr. Hyoscyami		
	Ext. Aspidospermæ (Quebracho)		
	Ext. Grindeliæ Robustæ	āā f ʒ i	4.00
	Syr. Pruni Virginianæ	q. s. ad f ℥ ii	60.00
	M.		

S.—One teaspoonful every three hours; for a child five years old.

A course of syrup of the iodide of iron with cod liver oil is very useful in all cases, and change of climate, to the seashore or inland, is sometimes effective in enhancing a permanent cure. In protracted cases a meat and milk-free diet may be tried. The patient is fed exclusively on well-cooked cereals—without milk or sugar—and vegetables. Sweet butter is added to make the food more palatable.

In treating asthma we should always bear in mind that asthma-like attacks are observed as a manifestation of a large thymus, spasmophilia, malaria, or heart and kidney disease, calling for specific therapeutic measures to remedy the underlying affections.

Emphysema Pulmonum

Abnormal distention of the lungs with air occurs as a result of forced inspiration, e. g., in stenosis of the larynx (croup) or bronchioles (asthma), whooping cough, in bronchitis or bronchopneumonia with violent coughing, etc., or from forcible expiration, e. g., cornet playing. Owing to the great elasticity of the puerile lung and its tendency to rapid adjustment, emphysema as a permanent affection is rarely observed in children. If it does occur, it is most frequently limited to the apices and the anterior borders of the lungs. Exceptionally the emphysema is disseminated throughout the entire lung. In this event the symptoms are practically the same as those in the adult—namely, exaggerated resonance on percussion, diminution of relative cardiac dulness, dyspnea,

fulness of the upper portion of thorax or barrel-shaped chest, and pro-
longed incomplete expiration. In cases of long standing there is con-
secutive involvement of the heart—usually dilatation of the right heart,
with or without hypertrophy.

The *treatment* consists, in addition to removal of the cause chiefly of
change of air (mountains), and light breathing exercises.

Bronchiectasis

Bronchial dilatation is not very uncommon in children, but as it
usually forms a sequel of respiratory diseases (unresolved pneumonia)
with violent coughing, or aspiration of foreign bodies into a bronchus,
its presence is frequently obscured by the symptomatology of the
preceding affection. Cases of congenital bronchiectasis are on record.

The dilatation of the bronchus may be cylindrical or sacculated,
and is almost always associated with peribronchial sclerosis (pul-
monary contraction), and occasionally with emphysema.

There are no pathognomonic signs of this affection, except, perhaps,
the copious morning expectoration of greenish-yellow, often fetid,
purulent mucus, which on standing separates into an upper layer of
serum and a lower of pus. Auscultation of the affected part of the
chest reveals abundant moist râles, and, if the bronchiectatic cavities
lie near the chest wall, cavernous signs, which greatly resemble those
of tuberculous cavities. In bronchiectasis, however, the sputum is
free from tubercle bacilli and the course is usually afebrile and often
remittent—the child often doing well for weeks. In cases of long
standing, there is usually clubbing of the fingers and deformity of the
chest. An extensive bronchiectasis may often be revealed by a roentgen-
ray picture.

Treatment.—Relative recoveries from this affection have been re-
ported particularly recently by surgeons who do not hesitate to per-
form pneumonectomy. Otherwise the majority of cases are incurable,
and after a shorter or longer (years) course the patients succumb
to intercurrent diseases, such as pneumonia, miliary tuberculosis, or
pulmonary gangrene.

The medical treatment is principally hygienic and prophylactic:
wholesome food, tonics, breathing exercises, inhalation of warm va-
pors with eucalyptus, creosote, or turpentine, or of oxygen, residence in
a high, dry region.

To facilitate emptying the dilated bronchi of their mucopurulent
content, gentle inversion of the little patient a few times a day proves
useful.

Pulmonary Gangrene

Gangrene of the lungs is not rarely a sequel of pneumonia, phthisis, grave exanthematous diseases, gangrenous processes of the mucous
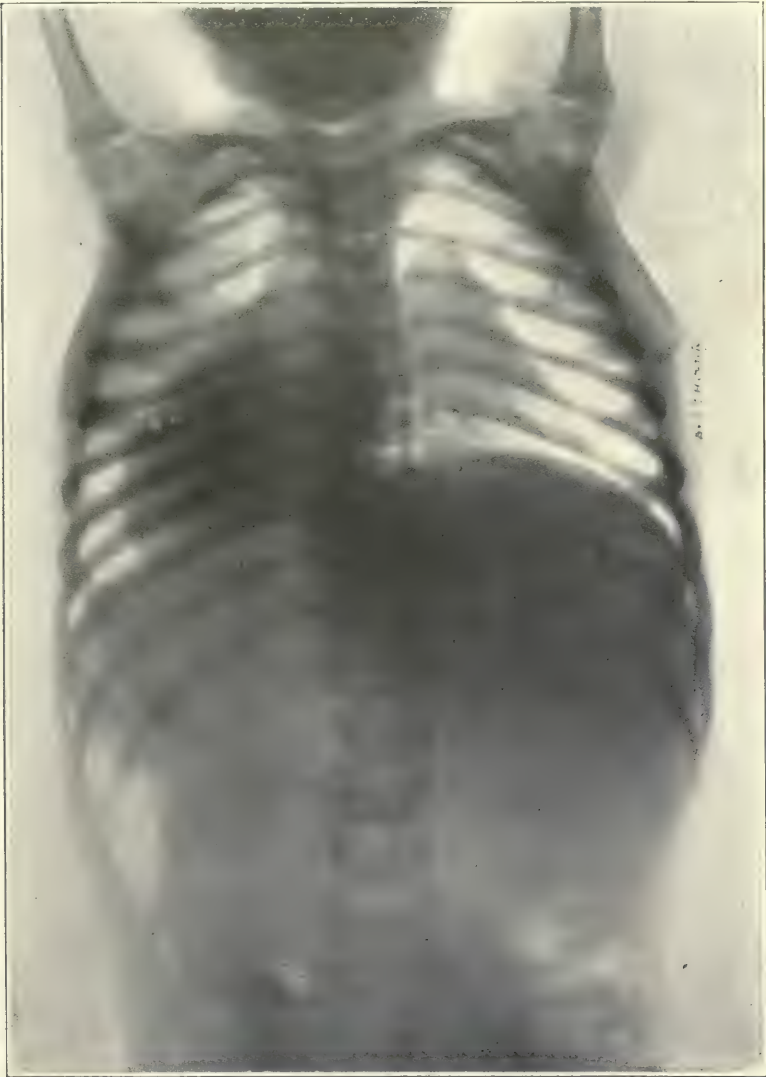

Fig. 79.—Pneumothorax (posterior view). Note compression of lungs and dislocation of heart.

membrane or of the skin, foreign bodies in the air passages (entrance of bits of food), etc. The symptomatology of this affection is ill

defined. In older children, as in adults, the macro- and micro-scopic appearances of the expectoration (upper layer, mucopurulent; middle, serous; lower, almost wholly of pus; remnants of lung tissue and plugs containing needles of fat, acids and detritus) are very helpful in the diagnosis. On the other hand, in infants, chief reliance must be placed upon the general cachectic condition of the patient, the coexistence of gangrene of the mouth, throat or vulva, the frequent occurrence of
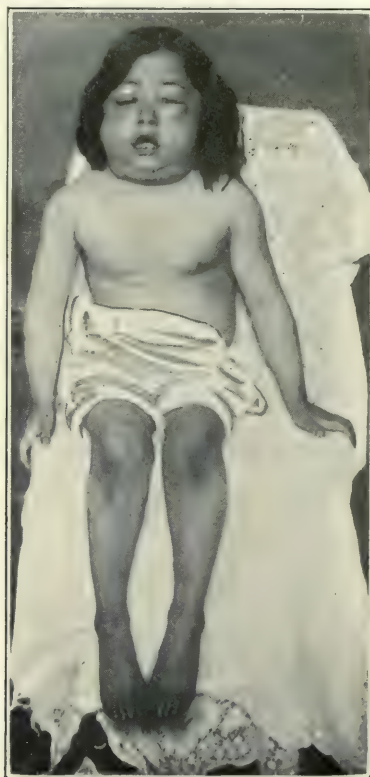
Fig. 80.—Pneumohypoderma (emphysema cutis) in a girl five years old complicating measles with pneumonia.

hemoptysis (absence of tubercle bacilli), fetid diarrhea, and foul breath. The cough is usually spasmodic.

The course of the disease is comparatively rapid, fatal termination usually occurring within a few weeks, either from gradual loss of strength or from complications, such as hemoptysis, pneumothorax, thrombosis, or cerebral abscess.

The *treatment* is symptomatic—tonics, inhalation of antiseptics, and, if the gangrenous process is accessible, surgical intervention.

Pneumothorax, Hemopneumothorax, Pyopneumothorax

These conditions occur principally as a result of traumatism (fracture of a rib or clavicle), laceration of the lungs from violent coughing or by foreign bodies, perforation of the lungs through empyema, gangrene and similar destructive processes.

The symptomatology is the same as in adults: sudden severe dyspnea, bulging of the affected side, tympanitic percussion sounds. When effusion occurs, there is hyperresonance over the upper portion of the

Fig. 81.—Same case as Fig. 80 six weeks later.

affected part of the chest above the line of effusion and dulness or flatness over the seat of effusion. Succussion gives rise to splashing sounds. The diagnosis can readily be corroborated by thoracentesis and Roentgenograms.

The *treatment* consists of the administration of opiates for the pain and aspiration (of air or fluid) to relieve the intense dyspnea in addition to attention to the primary cause.

Pneumohypoderma[*]

(EMPHYSEMA CUTIS)

Entrance of air into the subcutaneous areolar tissue ordinarily results from rupture or laceration of the pulmonary alveoli or bronchi during violent coughing or dyspnea (*e. g.*, in pertussis, measles, phthisis pulmonum), or secondarily to suppurative or caseous processes in the lungs. It is occasionally observed in connection with traumatic pneumothorax, and after tracheotomy and intubation. The air inflation may remain limited to the neck and face or spread over the entire upper half of the body, and exceptionally also to the lower half.

Pneumohypoderma can be detected by the distinct crackling or purring sensation imparted to the examining finger, and can readily be differentiated from anasarca by the absence of pitting on pressure. In severe cases the distention of the skin imparts to the palpating finger the sensation very much akin to that experienced when pressing upon a tensely inflated toy balloon.

If the immediate cause can be promptly arrested, *e. g.*, violent cough, by means of morphine, reabsorption of the air usually occurs within a few weeks. Rapidly fatal cases, however, are on record.

---

[*]The new term is suggested because it locates the exact seat of the trouble; it also helps to distinguish this condition from "surgical emphysema," which is produced by gasogenic bacteria.

CHAPTER VII

SPECIFIC COMMUNICABLE DISEASES

Influenza

(The Grip, The Flu, Spanish Influenza)

Influenza is an acute, highly communicable, endemic and epidemic disease, characterized by a variable group of respiratory, gastric and nervous phenomena, intense prostration and great tendency to grave complications and sequelæ.

Until the most recent epidemic, the bacillus of Pfeiffer was looked upon as the indisputable cause of this affection; since then, however, a great deal of evidence to the contrary has accumulated, which sheds doubt on its specificity. Col. Victor C. Vaughan, whose scientific and practical experience with the grip epidemic has been almost unlimited, does not hesitate to state that to him the evidence that the Pfeiffer bacillus as the cause of influenza is not at all convincing for the following reasons.* "In the first place, it is by no means constantly found in influenza or its sequelæ. In the second place, it is often even with greater frequency found in other diseases than it is in influenza. In the third place, influenza is characterized by a marked leucopenia, whereas injection of the Pfeiffer bacillus causes a leucocytosis—just the opposite!" Moreover, repeated experiments to communicate the disease by direct inoculation (subcutaneously, by the nose and throat and swallowing of influenza sputum) proved negative. On the other hand, some authorities maintain that the influenza bacillus is demonstrable in the majority of cases of influenza, but that it plays an unimportant part in the secondary, so often fatal, infections, the latter developing chiefly as the result of a characteristic violent reduction of the resisting power of the tissues, which offer the pneumococci, streptococci (hemolyticus and viridans), Friedlander bacillus, staphylococcus aureus and micrococcus catarrhalis a favorable culture medium to become markedly pathogenic.†

*Jour. Am. Med. Assn., Dec. 21, 1918.

†Bacteriologic examination of the pleural fluid removed surgically and sent to the laboratory together with that encountered at necropsy, revealed streptococci in most instances, occasionally *Staphylococcus aureus* and pneumococci. Bacteriologic examination of the pus in the intrapulmonary abscesses almost invariably yielded a pure growth of *Streptococcus hemolyticus*, but occasionally *Staphylococcus aureus*. In three cases, *Streptococcus hemolyticus* was isolated in pure culture from the blood. In the pneumonic exudates themselves, the prevailing microorganism was a streptococcus. In occasional instances, influenza bacilli and pneumococci were isolated in combination with one another or with streptococci. There were three cases in which

The *pathology* of the disease differs with every epidemic as well as with each individual attack. In cases of moderate severity the lining membrane of the rhinopharynx and lower portions of the respiratory tract are hyperemic and sparingly covered by a grayish, often very thick, deposit. The bronchi and bronchioles are filled with a mucopurulent secretion containing the aforementioned bacteria. Here and there the pulmonary alveoli are involved. In severe cases the inflammation extends throughout the entire lung and pleura. In the recent epidemic
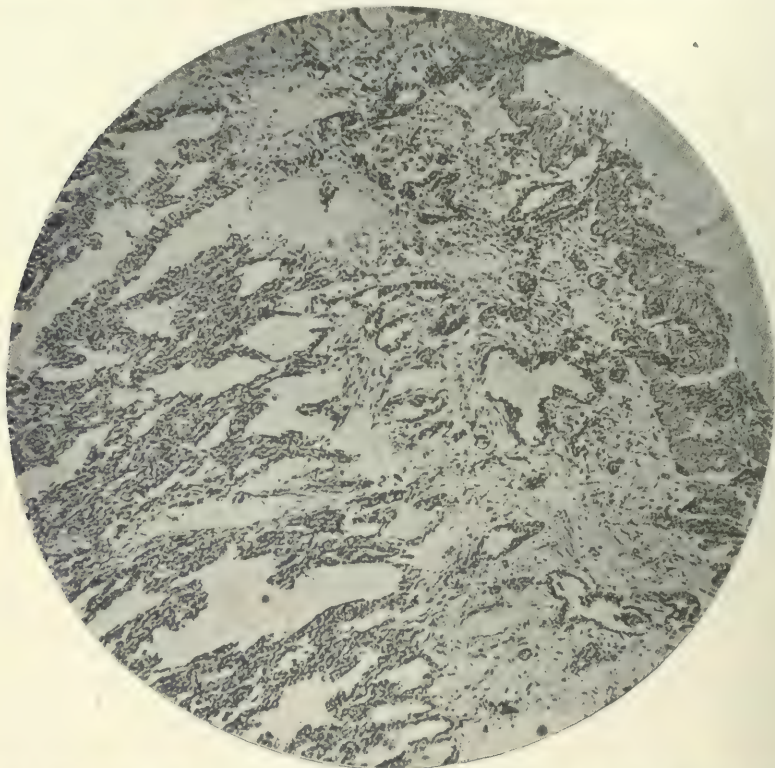
Fig. 82.—Section of lung of epidemic influenza in a young infant showing congestion of the blood vessels in the pleura and hemorrhages just beneath the pleural surface. (Drs. Martha Wollstein and A. Goldbloom.)

of the so-called Spanish influenza, Oberndorfer,* among many other clinicians and pathologists, found the following pathologic entity:

massive portions of a lobe were consolidated in such fashion as to resemble ordinary croupous pneumonia. In all of these the exudate was sticky. In two, *Bacillus mucosus-capsulatus* was isolated, in the other *Streptococcus mucosus*. In three other cases, streptococci were isolated from the blood during life; and all of them, at necropsy, presented abscesses of the lungs. (Symmers, Dimerstein and Frost, Jour. Am. Med. Assn., July, 1920.)

*Ueber die Pathologische Anatomie der Influenzaartigen Epidemie (München. med. Wchnschr., Vol. lxv, p. 810).

In the initial stage of the affection of the lungs, namely, when only small foci without any great reaction in the immediate neighborhood are observed, the most striking findings were small, bean-sized hemorrhages projecting into the lung tissue. As a next step there followed a firmer infiltration of the parenchyma, the nodules sitting subpleurally and raising the pleura in consequence. A whole scale of intermediate formations lay between these small nodules and large hemorrhagic tuberous infiltrations; all possible gradations were observed from simple blood extravasations into the lung tissue, still containing air, to firm, almost dry, infarct-like hemorrhages of a bluish-black tinge. These extensive infiltrations were of the same shape as the usual pulmonary hemorrhagic infarcts, namely, they had the form of a wedge with its base resting on the pleura, thus clearly indicating an intimate relationship with the vascular system of the lungs. In this purely hemorrhagic initial stage no thrombi were ever found in these arteries, the extravasation of red cells being obviously due to an abnormal permeability of certain portions of the arterial system.

The second stage was characterized by exudative pneumonic processes combined with hemorrhages. The picture varied considerably at times. There may be a true croupous hepatization of lobular, or even lobar, extent, both red and gray, though the tinge was usually brownish and not as a rule very distinct. These pneumonic infiltrations usually embraced in their center circumscribed hemorrhages. The surface on section was not dry, being covered by a slimy, dirty coating, thus resembling a picture of Friedländer's pneumonia. The pneumonic foci were sometimes flattened out at the ends into yellowish white wedge-shaped strictures corresponding to anemic infarcts both in form and color. These often became the seat of gangrene or suppuration, the pleura also being obviously involved in the process. In the majority of cases it was a catarrhal and desquamative succulent infiltration rather than a fibrinous exudation, but almost always severely complicated by suppuration.

The bronchi were filled with pus already in the first stage, the smaller branches containing thin fluid, though at times dried-up exudates formed firm plugs occluding the lumen of the bronchioli. This purulent bronchitis had as its consequence an extensive bronchiectasis with the bronchi distended cylindrically. The bronchi and their blood vessels were often surrounded by purulent infiltrations originating from the lymphatic system. In other cases, again, there were seen on section enormous numbers of minute abscesses surrounded by hemorrhages, the suppuration being obviously hematogenic in origin. These minute abscesses often became confluent, thus ending up by the formation of large caverns of pus.

The pleura participated in the process. The first signs consisted in punctiform hemorrhages, or ecchymoses; serous exudations followed next, and, as often as not, empyemas completed the picture. As a rule, one side only was affected. Pericarditis was a natural consequence of pleuritis. There were no gross changes in the heart save for some thickening of the arteries of the lung hilum. Occasionally incipient endocarditis was encountered.

The larynx and the upper third of the trachea showed no involvement in the process. The lower portion, however, was the seat of an intense mucopurulent exudation, which in many cases assumed a fibrinous character, with the consequent formation of extensive pseudomembranes in the lower trachea and down into the bronchi. Sometimes edema of the epiglottis was observed.

A striking feature was presented by the hyperplastic condition of the lymphatic apparatus of the tongue and the tracheal ring. The thymus was well preserved, the cervical and axillary, but not the inguinal, glands were enlarged.

In the postmortem examination of 18 infants, Drs. Wollstein and Goldbloom* found also subcapsular hemorrhages over the thymus, thyroid and suprarenals, and also small hemorrhages within the swollen bronchial and mediastinal lymph nodes.

The alimentary tract rarely escapes infection especially in young children. The spleen is enlarged, sometimes a septic spleen tumor being found. The liver is but seldom involved. The kidneys show a general hyperemia. In the brain and the meninges there is marked
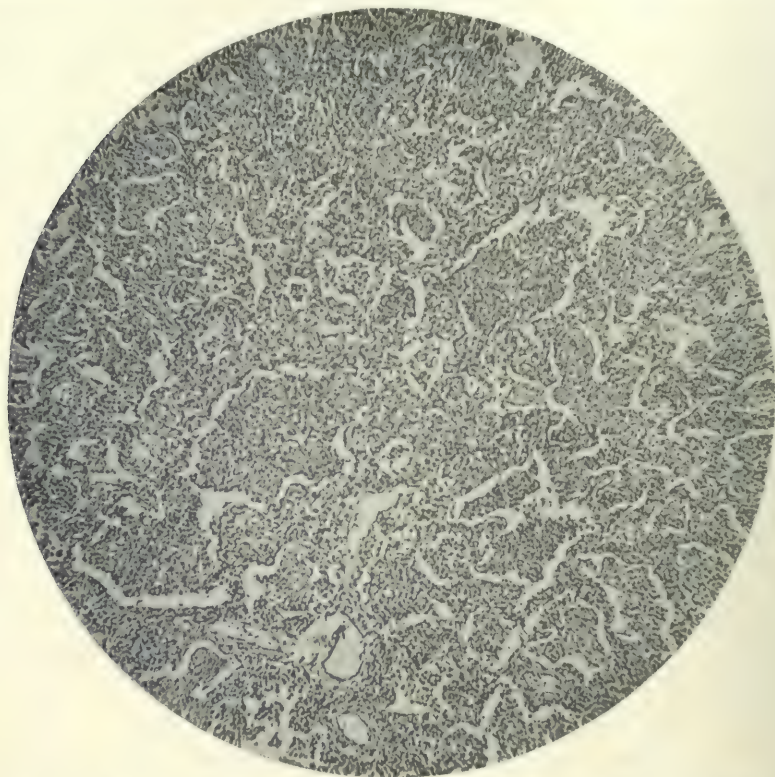

Fig. 83.—Section of lung of epidemic influenza in a young infant showing suppurative bronchitis and areas of pneumonia about the bronchi. The exudate is chiefly polynuclear in character. (Drs. Martha Wollstein and A. Goldbloom.)

vasodilatation, but no meningitis. Punctiform hemorrhages are met in different parts of the encephalon, and cases are on record in which the ventricles were filled with blood and pus. There are also hemorrhages in the heart valves, with a consequent displacement of the fibers and damage to the endothelium; thus, no definite endocarditis,

*Am. Jour. Dis. Children, March, 1919.

but lesions which facilitate the development of a secondary micotic endocarditis.

The essence of the whole pathologic picture consists, therefore, in the abundance of hemorrhages seen in the mucous and serous membranes, in the respiratory tract, and in the lungs, which indicate a damaged condition of the capillary vascular system. The whole process seems to be primarily a bacteremia localized in particular in the pulmonary blood vessels. From a purely anatomic point of view the condition bears many points of resemblance with pneumonic plague, though there is no indication as to an entry of the virus through definite lymphatic channels. Furthermore, as already stated, the true nature of the virus, the primary infecting agent, is still undiscovered.

Clinical Course.—No age is exempt from this affection, and one attack neither predisposes nor immunizes for any length of time against another one. The incubation period varies from two to five days. The onset is usually sudden or may be preceded by a few mild prodromata common to all febrile affections. The previous attempts to classify the grip into three distinct types,—namely, catarrhal, gastric, and nervous, was based upon an erroneous conception of the pathology of the disease. It is the multiplicity of the lesions and the complexity of the symptoms which are the characteristics of influenza. Thus, the child sneezes, coughs, has no appetite, vomits, complains of pain in the entire body, especially in the throat, head, eyes and lower extremities, has high fever, is very restless or lies exhausted in a semistupor —an indefinite group of symptoms which is met with in quite a number of acute febrile affections.

The onset is usually sudden, sometimes preceded by signs of fatigue, headache, and chilliness. This is followed by an abrupt rise of temperature up to 104° F. or higher, which usually continues during the entire course of the disease. The throat is deep red in color, and the tonsils and fauces are often covered with glairy mucus and occasionally with a yellowish-white irregular deposit. The cough is dry, harsh and painful, especially over the region of the sternum, and large, soft or dry sibilant râles are heard over the greater portion of the thorax. In infants particularly, there are more or less pronounced manifestations also of the alimentary tract. The baby vomits, refuses food, cries from abdominal pain, and has an increased number of foul smelling, variously colored, thin evacuations. In older children the gastrointestinal symptoms are usually limited to anorexia, tympanites and occasionally constipation. The nervous system is almost invariably implicated in the grippel process. Among the characteristic nervous phenomena we may mention, in the order of their frequency, hyper-

esthesia, headache, somnolence, insomnia, vertigo, and convulsions. The child cries when it is being lifted or moved about in bed. The pain in the head, neck, trunk and extremities often keeps the little patient in a position closely resembling opisthotonos, and if accompanied by convulsions, one is often tempted to diagnose the symptom complex as meningitis. The somnolence is frequently profound. One baby under observation dozed for six days, awakening with a fit of crying when disturbed even for nursing. On the other hand, some children keep awake for several days in succession, notwithstanding the administration of hypnotics. Vertigo in infants usually escapes our notice; in those able to hold up their heads, it is manifested by the latter dropping forward or swaying in different directions. The eyelids
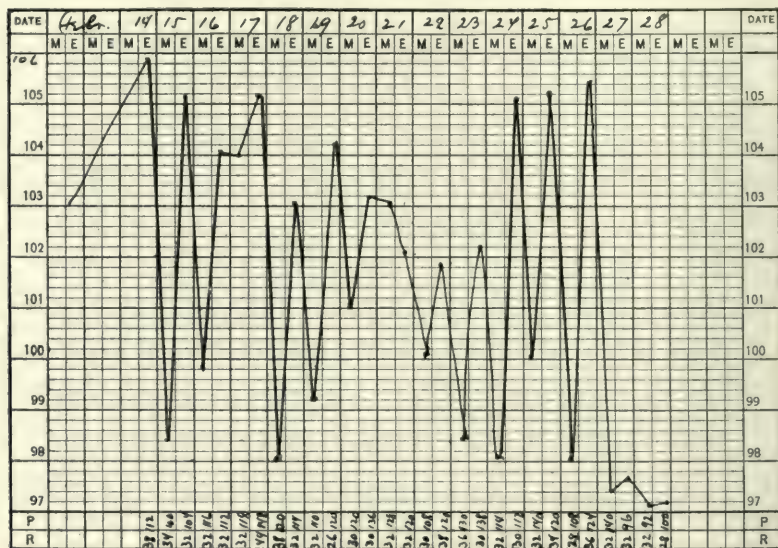
Fig. 84.—Fever curve of atypical influenza in a baby fourteen months old.

droop, the face turns pale and vomiting supervenes. Older children invariably complain of this miserable feeling, and find difficulty in holding their heads erect.

The blood shows a striking leucopenia, even in the presence of complicating pneumonia. According to L. A. Conner, a leucopenia of from 2,000 to 3,000 white blood cells per cubic millimeter is by no means uncommon. Leukocyte counts were made by Montgomery and Dunham* in thirty cases of influenza occurring in infants and children less than 12 years of age. They found that the tendency in uncomplicated in-

*Am. Jour. Dis. Child. Vol. 18, No. 3.

fluenza in infants and children is toward a leukopenia, rather than a leukocytosis. There is a tendency to a slight leukocytosis in complicating pneumonia. In this series, in all pneumonia cases resulting fatally, the leukocyte counts were under 10,000. The prognosis, in general, is better in pneumonia cases which exhibit a leukocytosis. Differential counts have shown: (*a*) a tremendous variation in the

Fig. 85.—Paralysis of N. abducens, with convergent strabismus and facial paralysis following postinfluenzal encephalitis. Her mentality remained greatly affected.

differential formula, and (*b*) nothing sufficiently constant to be of clinical aid in diagnosis or prognosis.

Influenza shows a peculiar predilection for hemorrhagic processes, such as epistaxis, hemorrhage from the bowels, in the skin, the ears and pleural cavity, and in the urinary tract. In a contribution to the study of this disease (N. Y. Med. Jour., June 30, 1900), I called attention to the concurrence of hemorrhagic encephalitis during the course of the grip, and have since had occasion to observe several such cases. This form of encephalitis seems to be identical with the so-called leth-

DISEASES OF CHILDREN

argic encephalitis (see p. 624) so frequently noted during the recent influenza epidemic and thereafter. One boy, eight years old, under my care, died from this complication twelve hours after its onset. As a rule, the mortality in these cases is rather low. Of 8 cases recorded by H. Heiman[1] none died but 2 of them remained imbeciles. Similar cases were recorded by me[2] several years ago.

In uncomplicated sporadic cases the tendency of influenza is toward rapid convalescence and recovery, especially in strong children and those free from hereditary and acquired encumbrances. Unfortunately, however, influenza, by reducing the power of resistance of the patient, predisposes to prolific, often fatal, complications. In some epidemics ear affections predominate, in others cerebrospinal disorders, and in others again, as it happened in the last epidemic, respiratory affections devastate the world. The so-called "influenza pneumonia" usually sets in the second or third day of the disease, at a time when the influenza proper seems on the wane. The patient, especially if an infant, begins to vomit again, and shows definite signs of being ill at ease, without being able to locate the seat of his discomfort. The cough loosens, but the dyspnea increases and the heart beat, as a rule, slows down and gives the impression of being more steady in quality. At this stage careful physical examination of the thorax often utterly fails to elicit any signs of pneumonia, except, perhaps, exaggerated breathing over the posterior portions of the lower lobes. On many occasions I was most painfully surprised to find what at first seemed to be a simple bronchitis, within but a few hours transformed into double fatal pneumonia. Similar to erysipelas of the skin, the inflammatory process of influenza seems rapidly to spread by contiguity from structure to structure and from one organ to the other, so that on every examination of the patient a newly diseased focus is detected. Cyanosis,* delirium and coma generally precede the fatal issue, which usually takes place within three to five days. Those children who withstand the exhausting effects of the violent acute stage often survive, even though convalescence and recovery may very frequently be markedly delayed by additional grave complications and sequelæ. As already stated, the pleura very rarely escapes involvement. The pleuritic effusion is not infrequently hemorrhagic or purulent in character from the start and accumulates in the thorax with extraordinary rapidity. In an infant ten months old the left side of the chest was filled with pus on the sixth day after

[1] Am. Jour. Dis. Child., August, 1919.
[2] H. B. Sheffield, "The Backward Baby," p. 72.

*The cyanosis is caused by an abnormally high oxygen unsaturation of the blood, which may be produced either by an admixture of reduced hemoglobin and oxyhemoglobin in the superficial capillaries, or by an incomplete oxidation of the venous blood in the lungs. (W. C. Stadie, New York, and C. Lundsgaard, Copenhagen. Jour. Exp. Med., Vol. 30, No. 3.)

the onset of the influenza. Otitis is very common during the early catarrhal stage of the disease. In the majority of instances the inflammation clears up without suppuration, but as already mentioned, in some epidemics the ear infection is most virulent, sometimes terminating in mastoiditis simultaneously with the early appearance of congestion of the tympanum. Nephritis (often hemorhagic) and pyelocystitis form an early or late complication. In malignant cases the peritoneum, pericardium and meninges are attacked in rapid succession, in fact, as we are here dealing with a general bacteremia, no organ of the body is spared; hence it is of little consequence, from a therapeutic point of view, where the infection begins and where it ends. Among the milder complications and sequelæ we may mention cardiac neuroses (bradycardia and tachycardia), neuralgia, neuritis, arthritis, adenitis, parotitis, conjunctivitis, inflammation of the accessory sinuses, and occasionally periostitis. Similar to measles, influenza predisposes to tuberculosis, and in a number of instances divers psychoses were noted to follow the disease, sometimes several weeks after apparent recovery.

Finally, every form of cutaneous eruption may accompany influenza. At times the eruption is bright red and punctiform, resembling that of scarlatina, or roseolar or pustular in character which may readily be mistaken for rubeola or varicella. Urticaria is not uncommon, and simple erythema may be seen in the majority of cases at some stage of the affection. The peculiar facial flush ("lobster face") often noted in adults, is very rarely observed in children. The hemorrhages in the skin may assume the appearance of purpura hemorrhagica.

The *diagnosis* of influenza is comparatively easy during its prevalence in epidemic form, but quite the reverse otherwise. "Colds" and gastrointestinal disorders being of such ordinary occurrence in children that influenza is generally not thought of when such manifestations present themselves. Furthermore, the diagnosis is often obscured by the numerous complications. For general guidance in the diagnosis we may state that the simultaneous development of respiratory, digestive and nervous phenomena, leucopenia and marked prostration should always arouse our suspicion, even in the absence of an epidemic of influenza.

As already emphasized the *prognosis* varies greatly with each epidemic, and the mortality may range anywhere between 10 and 30 per cent. Of course, the outcome of the disease depends entirely upon the number and severity of the complications.

Treatment.—Influenza is always to be looked upon as a treacherous and dangerous disease, however mild its appearance in its early stages;

and appreciating the fact that it is so highly communicable and so grave in its consequences, it is obvious that every effort must be made to arrest the disease at its source by strict isolation of the patient, and to employ every means to prevent the grave complications and sequelæ. During the recent epidemic an attempt was made to attain these objects in view by prophylactic vaccination. In order to avoid burdensome repetition and controversies of different clinicians, we shall take the liberty of citing the views of G. W. McCoy,[1] Director, Hygienic Laboratory, U. S. Public Health Service, which embody the consensus of opinion of the profession as a whole.

Vaccine from Influenza Bacillus as a Prophylactic

In discussing this subject, we will give attention, first, to the results obtained from the use of a vaccine made from the influenza bacillus alone, or from other suspected etiologic agent, which aims, to be sure, to prevent the primary disease, and later to a review of the evidence with respect to vaccines which have been devised with the special object of preventing the development of pneumonia or of mitigating its severity.

A vaccine made from the influenza bacillus alone seems not to have appealed sufficiently to European workers to induce them to try it when the epidemic prevailed abroad. In this country, its use has been confined largely to New England. The early reports on this vaccine were very encouraging; figures were presented which, if taken at their face value, would convince any one of the efficiency of the agent; but, when these figures were submitted to careful analysis, much doubt remained as to whether the vaccine was of any service whatsoever. The chief source of error lay in the fact that the inoculations had been done during the progress of the epidemic, and that the case incidence among the vaccinated was compared with the case incidence in the general population or in the control groups from the beginning of the epidemic. Now, it is plain that if, after the epidemic is well under way, we vaccinate a portion of the persons in a population, the percentage of persons attacked will be smaller among the vaccinated than among the nonvaccinated, because a percentage of the total number of cases will have occurred before the vaccine is given. Not only does this introduce an error by counting in the control, or nonvaccinated group, cases that have occurred. early, but also it leaves a select group to be vaccinated, wholly or in part, in which the percentage of susceptibles is smaller than in the original group of which they formed a part.

To make this clear, let us suppose that ten days after an epidemic started in a population of 1,000 persons, an admittedly worthless vaccine was administered to one half of those who at that time remained unattacked by the disease. Let us further assume that on the date of vaccination 20 per cent of the population had sickened, leaving 800 well persons, of whom 400 were vaccinated. Since the hypothetical vaccine is worthless, the morbidity will be as great in the vaccinated as in the nonvaccinated group. Let us assume this to be an additional 20 per cent. Then the total morbidity in the vaccinated group will be 20 per cent of 400, or eighty cases. The total morbidity in the unvaccinated, however, if we consider

---

[1] Jour. A. M. A., Aug. 9, 1919.

the entire period of the epidemic will be 20 per cent of 1,000, or 200, plus 20 per cent of 400, or eighty, which would make 280 cases.

Although the error is now sufficiently clear, we have seen reports which, on the basis of the above figures, if applied to this hypothetical worthless vaccine, make it appear to be a valuable prophylactic. The statement of these reports would be, in effect, that one half of the population was vaccinated, that among the vaccinated only eighty cases developed, while among the unvaccinated 280 cases appeared. Hence the obvious value of the vaccine.

We must also remember that a vaccine can scarcely be expected to exert any appreciable prophylactic effect before from seven to ten days after the vaccine is given, since a week or more is required for immunity to develop. A comparison is fair which considers, among both vaccinated and nonvaccinated, only cases that have occurred, say, ten days or more after the vaccinations are made.

When the influenza bacillus vaccine was submitted to such critical tests as the inoculation of approximately half of the individuals in institutions, or in other large groups, its failure became apparent. A few examples of this are worth citing. Hinton and Kane were able to vaccinate about half of the patients at an epileptic colony long enough before the disease became prevalent in the institution to justify the drawing of conclusions from their data. The vaccine used contained 800,000,-000 organisms per mil, and a total of 2,000,000,000 were administered to each person. The results were as shown in Table 1.

TABLE 1.—EFFECT OF INFLUENZA BACILLUS VACCINE AS A PROPHYLACTIC

	VACCINATED		NOT VACCINATED (CONTROLS)	
	NO.	PER CENT	NO.	PER CENT
Number of persons	461	....	518	....
Cases of influenza	163	35.4	178	34.3
Deaths	28	17.0	24	13.5

On the basis of this experiment the authors reach the obvious conclusion that the vaccine was without value.

A similar test was made on the naval personnel at Felham Bay Training Station; here a part of the individuals of a group were vaccinated, the remainder being held as controls. According to the latest available report, 9 per cent of the 554 inoculated persons developed the disease, and 5 per cent of the 800 who had not been inoculated developed it.

Similar failure attended the attempts at immunization of men at the naval base at Paris Island, S. C. It was definitely shown that neither incidence nor severity was influenced by the vaccination. These observations were all on groups large enough to make the deductions of value.

A number of controlled vaccinations, in which influenza bacillus vaccine was used, carried out in institutions by the Public Health Service, gave the rather para-doxical result of showing an increased percentage of attacks among the vaccinated, but more deaths among the nonvaccinated. This result was obtained with a vaccine directed against the primary disease, not against the complicating pneumonia. The results are shown in Table 2. These figures illustrate the fallacy of giving much weight to the results of a small set of observations in work of this sort.

TABLE 2.—RESULTS OBTAINED BY INFLUENZA BACILLUS VACCINE IN INSTITUTIONS

| | VACCINATED | | NOT VACCINATED (CONTROLS) | |
	NO.	PER CENT	NO.	PER CENT
Number of persons	484	....	842	....
Cases of influenza	153	31.6	223	26.4
Deaths ....,.........	0	....	4	1.8

VACCINES FROM STREPTOCOCCUS AND OTHER ORGANISMS

Another series of vaccinations aimed directly against the supposed causative agent was that reported by Ely, Lloyd, Hitchcock and Nickson. These workers believed that the epidemic was due primarily to a hemolytic streptococcus which could be detected in the blood and in the lungs. From the fact that the organisms with which these observers worked soon lost their chain-forming properties and, in some instances, the power to hemolyze promptly, they express some doubt as to whether they should be classed as streptococci, and they further assume that there are material differences between different strains. The results of the use of a vaccine prepared from organisms isolated from the cases were apparently most encouraging, though none of the experiments was controlled in a manner that would definitely establish the value of the preparation. The work of these observers needs to be repeated before the results can be accepted for general application.

When we come to consider the evidence with respect to the vaccines especially designed to prevent the pneumonic complications of influenza, we find again such conflicting reports that one is somewhat bewildered.

The only papers from a foreign source that have come to my notice are those by Eyre and Lowe, who used a mixed vaccine which contained the pneumococcus, the streptococcus, the influenza bacillus, *Staphylococcus aureus*, *Micrococcus catarrhalis*, *B. pneumoniæ* and *B. septus*.

These authors believe, and indeed present rather convincing figures in their first paper to prove their point, that the use of this vaccine produces lowered resistance, which may last for "from a few hours to two or three weeks," during which period the incidence of respiratory infections would be increased among inoculated groups.

The early experience of the English authors does not refer directly to the prophylaxis of influenza, but it is cited here to show that there may be an element of danger in the indiscriminate use of vaccines in the presence of a rapidly spreading epidemic like influenza in which naturally many persons in the "negative phase" would be attacked.

In a later paper, the same writers report on the experience with vaccine in the epidemic in England in the autumn of 1918. Stress is laid on the necessity of preparing a vaccine from cultures but recently isolated.

The figures given and the facts presented by these writers are difficult of interpretation and permit of almost any conclusion that one wishes to draw from them, from the optimistic one that fatalities after influenza occur only among the non-vaccinated, to the pessimistic one that fatalities occur only among the vaccinated, though the authors believe the results were good. They frankly reiterate the opinion that for a short time following vaccination there is an increased incidence among the vaccinated, owing to temporarily increased susceptibility, but the writers consider that this risk is justified by the benefit that they believe may accrue later. As inoculations were performed largely during the prevalence of the epidemic, and as

the controls appear to include persons who developed the disease prior to the vaccination, the alleged good results may be misleading.

THE POLYVALENT VACCINE OF ROSENOW

Rosenow prepared a mixed, and, at least in part, polyvalent, vaccine from the various fixed types of pneumococci, pneumococci of Group IV, hemolytic streptococci, Staphylococcus aureus and the influenza bacillus, all of which had been recently isolated. This vaccine was adjusted to meet the bacterial flora encountered during the epidemic; thus, in a manner it may be said that it was designed to approach an autogenous vaccine, but was intended primarily for prophylactic purposes. Dr. Rosenow felt that this vaccine should be prepared for use in any community from the strains of organisms there prevailing, and that a vaccine adjusted to meet the needs of one locality might not meet those of another. The figures given for protection are encouraging, but do not lend themselves to critical analysis.

Vaccine prepared in the manner suggested by Dr. Rosenow should theoretically have a better chance for success than those we shall next consider, but the practical difficulties of preparing it from locally prevailing strains and adjusting it to meet the changing flora of the respiratory tract in a disease that spreads as rapidly as influenza are obvious.

A specimen of the vaccine which was being used in Illinois was tried in California, under rigidly controlled conditions, without success. The disease did not appear in the institution where the test was made until eleven days after the last injection, but, after the epidemic had swept through it, the results revealed that 37 per cent of the vaccinated were attacked, against 28 per cent of the controls, while 4.5 per cent of the vaccinated population died, against 3.6 per cent of the nonvaccinated. These are differences too small to be significant. Tests made in other institutions gave similar results, though we need not take the time to consider the details here.

The only report we have on a vaccine directed against the influenzal pneumonias associated with the fixed types of the pneumococcus is that of Cecil and Vaughan, whose work was conducted at Camp Wheeler and was directed primarily against the usual pneumonias of the camp. Apparently the antipneumococcus vaccine reduced somewhat the incidence of influenzal pneumonia among the vaccinated, though, to use the author's words, ''influenza causes a marked reduction in resistance to pneumonia even among vaccinated men.'' These authors show clearly that the case mortality of secondary pneumonias was not reduced by the vaccination, contrary to the claim so often made, that the vaccine, when it fails to protect perfectly, at least leads to a milder type of the disease. Cecil and Vaughan believe that the results of their experiment with respect to pneumococcus pneumonia were obscured by the influenza epidemic; evidence that the prophylactic action of the vaccine employed against influenza was not striking, since the epidemic should have served to emphasize rather than obscure the results of the beneficial action of a really valuable prophylactic agent.

The general consensus of opinion of the profession seems inclined to the belief that prophylactic and therapeutic vaccination has failed in a definite manner to influence either the morbidity or the mortality of influenza.

Until a specific vaccine or drug against influenza will be perfected, we will be obliged to treat it symptomatically: the salicylates, with

or without quinine and phenacetin, for the relief of temperature and pain; mild expectorants, with or without small doses of codeine, to allay the cough; heart tonics, especially digitalis, to sustain the heart's action; hydrotherapy in the form of cool sponging or warm baths for hyperpyrexia; oxygen for the dyspnea and cyanosis; lumbar puncture for delirium and convulsions, and absolute rest in bed and a light diet, to maintain the patient's vitality. The nose, mouth and throat should be kept clean by means of Dobell's solution, and the alimentary tract free from putrefactive matter by a daily intestinal irrigation and mild laxatives. Complications arising should be treated according to indications. (See "Pneumonia," "Nephritis," "Peritonitis," "Encephalitis," etc.)

℞	Acid. Acet. Salicyl.	gr. xv	1.00
	Caffeinæ Natrii Benz.	gr. v	0.3
	Chocolate et Sacchari q. s.		
	M.		
	Div. in pulv. No. viii.		

S.—One powder every three hours, for a child three years old.

℞	Natrii Salicyl.	ℨ ss	2.0
	Potassii Citratis	ℨ i	4.0
	Extr. Glycyrrhizæ Fl.	ℨ ii	8.0
	Aq. Anisi q. s. ad	f ℥ ii	60.0
	M.		

S.—One teaspoonful every three hours, for a child three years old.

℞	Liq. Ammonii Anis.	ℨ ss	2.0
	Natrii Benzoici	ℨ ss	2.0
	Syr. Ipecacuanhæ	ℨ i	4.0
	Tr. Digitalis	ℨ ss	2.0
	Syr. Altheæ	℥ i	30.0
	Aq. Anisi q. s. ad	f ℥ ii	60.0
	M.		

S.—One teaspoonful every four hours, for a child three years old. One-twentieth of a grain of codeine may be added to each dose of the above medicine, if the cough is distressing.

Rubeola

(Morbilli, Measles)

Measles is probably the most frequent and most readily communicable eruptive fever of childhood. Children of from two to six years are most susceptible to it, but it is not rarely met with in older and younger ones, and cases in the newborn have been reported. In the majority of

instances one attack immunizes the patient against another one, numer-
ous exceptions, however, are on record. The cases of recurrent measles
often prove to be rubeola on one occasion, and rubella, or a similar skin
eruption, on another. The disease is communicable in all its stages
(particularly the catarrhal stage) by means of the as yet unknown con-
tagium*—which dwells in the lacrimal, nasal, and bronchial secretions,
and probably also in the papules and squamæ—either by direct contact
or, more rarely, through intermediate persons, the air, or fomites.

Nine to fifteen days—the period of incubation—pass after invasion of
the system by the materia morbi without any characteristic manifesta-
tion of ill health, except slight anorexia, restlessness, ephemeral rise
of temperature, etc., which toward the end lead to a more acute aggra-
vation of the condition and mark the beginning of the prodromic stage.
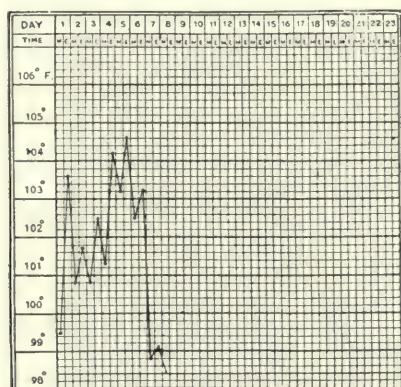
Fig. 86.—Fever curve of measles.

This stage usually lasts three days, rarely longer (up to a week in de-
bilitated children). The little patient complains of chilliness, headache,
and fatigue, hangs its head or sleeps most of the time, coughs and occa-
sionally sneezes, and presents a .rise of temperature of from 2° to 4° F.
Not rarely the fever drops the next day, but the catarrhal symptoms
continue in severer form. Examination of the mouth and throat in the
majority of cases reveals upon the mucous membranes of the soft and
hard palate diffuse redness or punctiform or stellate spots, and on the
buccal mucous membrane and inner surface of the lips from six to
twenty, rarely more, red spots, with a central, rounded, slightly elevated
bluish efflorescence. These spots never cause pain or ulcerate. They
are called Koplik spots—the latter deserving the credit of having proved

*R. Tunnicliff (Jour. A. M. A., April 7, 1917) has discovered a coccus in the blood and in
the nose and throat in the very early stages of measles. Whether this coccus is the primary
cause or a secondary invader is still undetermined. Measles has recently been produced ex-
perimentally.

the pathognomonic significance of the spots as an early sign of measles. This enanthema of the buccal mucous membrane not rarely appears from three to five days before the exanthema.

Another twenty-four hours and the eruptive stage is reached. Bright red, pinhead- to lentil-sized dots appear over the forehead, about the ears and over the face (chin and around the nose and mouth—circumoral ring), and rapidly enlarge to irregularly serrated, pea- and bean-sized, sharply circumscribed, rounded or crescentic, slightly elevated red spots, which disappear on pressure. From these points the eruption rapidly spreads, often in crops, over the body and limbs, taking about twenty-four hours to complete the process. At this time the catarrhal symptoms also are at their height. The face is flushed and edematous, the eyes are red and watering and dread light; the nasal catarrh is intense, the cough frequent, harsh and often barking, the voice hoarse, the temperature high (104° F., or higher), the urine scanty and high colored (diazo reaction often positive); the child is drowsy, at times delirious, often vomits and occasionally suffers from diarrhea (sometimes bloody, especially during the hot summer months). The peripheral and lymphatic glands are not rarely swollen and painful, and the spleen is somewhat enlarged.

It is generally accepted that cases of measles in which the exanthema appears on the back first are usually grave in character. The same holds true of the cases in which the exanthema suddenly fades.

The eruptive stage lasts from five to six days. Toward the end of the stage the eruption begins to fade, especially on the face, and bran-like scales take the place of the exanthema. With the fading of the eruption there is often a critical decline of the temperature (sometimes preceded by morning or evening remissions) and its concomitant symptoms, except the bronchial catarrh. The desquamative stage lasts about one week, so that the patient is usually entirely well by the end of the fourth week from the time of infection. Sometimes traces of the exanthema in the form of bluish-red spots remain over some portions of or the whole body which do not disappear on pressure with the finger. They are of no special significance.

Deviations from the typical course of the disease are not rare. Thus, the exanthema may be absent or so scanty as to escape observation— *morbilli sine exanthema*—notwithstanding the pronounced character of the catarrhal and febrile symptoms. In such cases the diagnosis from the grip is almost next to impossible, and can at best only be surmised in the presence of an epidemic or another case of measles in the immediate surroundings.

The eruption may appear in the form of small papules, at times pene-
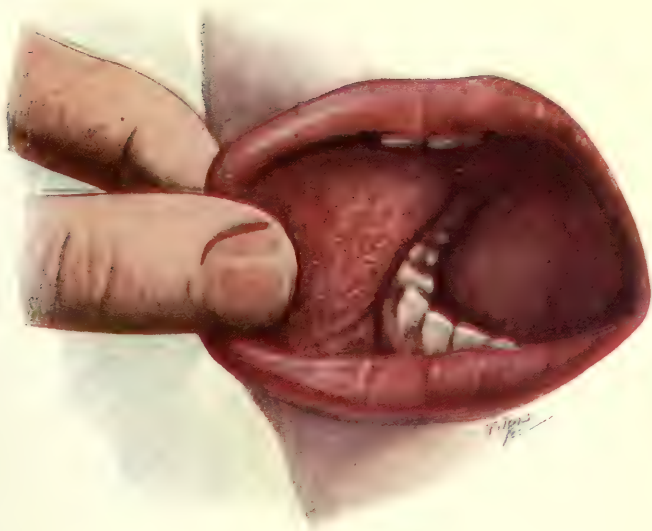
PLATE VI

BUCCAL ENANTHEMA IN MEASLES (KOPLIK'S SPOTS)

(Courtesy of Dr. John Zahorsky.)

trated by a hair—*morbilli papulosi;* or be covered by minute vesicles—*morbilli miliares.*

The appearance of the exanthema may be delayed for a day or two and then be localized principally upon the body and limbs or become confluent so as to resemble the rash of scarlatina—*morbilli scarlatinosi.* Occasionally small hemorrhages occur between the spots—*morbilli hemorrhagici.* This form of measles is not to be mistaken for *morbilli hemorrhagici maligni,* "black measles," which is rather very rare and observed only in delicate, cachectic children. In this condition instead of the eruption there are numerous petechiæ and ecchymoses, in addition to hemorrhages from the nose, ears, genitalia, kidneys or bowels. Malignant measles is usually associated with early depression, very high temperature, rapid and frequent pulse, dry, brown and thickly coated tongue, sopor, convulsions and coma, and often ends fatally within three days.

Occasionally the temperature is protracted or after a fall suddenly rises, indicating the occurrence or near advent of complications or sequelæ. Ordinarily complications set in toward the end of the eruptive stage, but may appear as early as the prodromic stage. At this period also we are apt to find angina tonsillaris, epistaxis, severe vomiting and diarrhea, catarrhal laryngitis, pneumonia, etc.

In the eruptive stage pneumonia forms the chief complication. Violent coughing is prone to give rise to laceration of the lungs and consecutive "pneumohypoderma." (See p. 344.) Quite frequently we meet also with pseudocroup and more rarely with diphtheria. The diphtheria of the throat sometimes develops secondarily to that of the conjunctiva; more frequently, however, the former occurs primarily, and the diphtheritic conjunctivitis remains limited to the original focus. It was my privilege to see 2 cases in point. One boy, six years old, succumbed to laryngeal diphtheria complicating measles, while his brother, three years old, was saved from blindness, and perhaps death, by early administration of antitoxin. The affected eye presented a clinical picture resembling that of gonorrheal ophthalmia. The diphtheritic conjunctivitis cleared up entirely withn ten days, but was followed by typeal diphtheritic paralysis of the throat. Severe stomatitis is not uncommon, and numerous cases of noma (*q. v.*) complicating or following measles are on record. The same observation holds good for divers forms of ear affections. Measles is not infrequently associated with typhoid, erysipelas, varicella, scarlatina, and acute pemphigus. The latter eruption may become gangrenous and prove fatal. The tendency to gangrene of apparently mild lesions of the mucous membranes and skin should always be borne in mind, as it is not at

all rare to find general sepsis supervening just such lesions. Measles acts as a great predisposing cause to pertussis, which latter may prove very serious owing to early supervention of bronchopneumonia. Sudden heart paralysis is rare.

Among the sequelæ the following affections deserve special emphasis: otitis, chronic conjunctivitis, keratitis, deafness, deafmutism, osteomyelitis, purulent pleurisy or pericarditis, nephritis, chronic bronchopneumonia, psychoses, meningitides and other nerve affections.

Measles manifests also a great disposition towards pulmonary tuberculosis (from 5 to 15 per cent of cases in some epidemics). As distinguished from scarlatina the blood in measles shows a subnormal number of leucocytes or a leucopenia.

Fortunately, most of the aforementioned complications and sequelæ are rare. Ordinarily, measles runs a benign course. Still, measles should always be looked upon as a very serious disease, especially if it attacks very young and delicate children and those with a tainted hereditary disposition. Indeed, in such children, especially if housed in asylums or hospitals, the mortality may vary from 20 to 40 per cent.

Treatment.—The custom still prevailing with some ignorant people to congregate the children free from measles with those affected by it, so that "they should all have it at once" is condemnable. Isolation of the patient should be insisted upon, and all other precautions available strictly adhered to. (See p. 69.)

The use of convalescent serum as a preventive and curative of measles has lately engaged the attention of pediatrists. The results are still sub judice. The usual measures in the treatment of measles consist principally of active diaphoresis by hot drinks, hot baths, and diaphoretics (decoction of crocus one dram to ½ pint), and minute doses of an opiate and expectorants to relieve and loosen the cough. Attention to complications is all important, whether grave or mild. A light diet should be enforced as long as the temperature is above normal. The fear of free ventilation of the sick-room is unfounded. On the contrary, a liberal supply of fresh air (68° to 70° F.) should be allowed as a therapeutic measure. Where photophobia exists, the room should be darkened by shades.

The mouth and eyes should be kept clean with warm boracic acid solutions, and the nasopharynx by instillations of a few drops of albolene. The temperature and nerve irritability should be reduced by small doses of phenacetin (½ grain for every year of the child's age) as well as by warm baths or packs (90° F.).

Other symptoms should be treated according to indications.

R̵	Liq. Ammonii Anisat.	3 ss	2.0
	Potassii Citratis		
	Syr. Ipecacuanhæ	āā 3 i	4.0
	Syr. Picis	3 iv	15.0
	Aq. Anisi	q. s. f ℥ ii	60.0
	M.		

S.—One teaspoonful every three hours, for a
child four years old (useful diuretic, diaphoretic
and expectorant). A small dose of codeine or
heroin may be added, if the cough interferes
with the child's rest.

For differential diagnosis see p. 398.

Rubella

(Rötheln, German Measles, Epidemic Roseola)

On superficial examination rötheln closely resembles measles, but
on careful observation it is found to differ from it in so many respects
as to justify its classification into a distinct disease. It is highly
communicable and often occurs in epidemics. One attack confers but
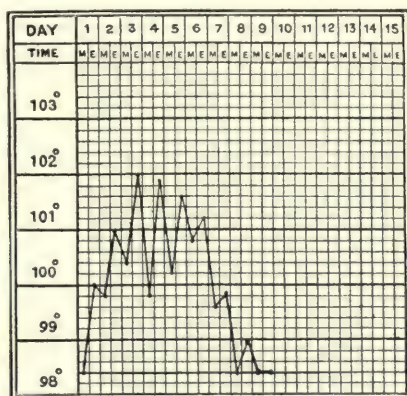
Fig. 87.—Fever curve of German measles.

little immunity against another, and not at all against genuine measles.
The incubation period lasts from ten to twenty-one days, and is gen-
erally free from any manifestations. There are none or very slight
prodromata of from twenty-four to forty-eight hours' duration, con-
sisting of languor, anorexia, and slight catarrhal symptoms, such as
mild injection of the conjunctiva, short cough and slight rhinitis. The
eruption usually appears suddenly, first on the face, and within from
twelve to twenty-four hours over the entire body. Often it has dis-

appeared from the face by the time the extremities are involved. The rash appears in *two* forms. One resembles that of measles—pale, red papules, up to the size of a lentil, usually discrete, rarely confluent, and momentarily disappearing on pressure. The other form is finely punctate, and coalesces into diffuse rose-red patches—resembling the rash of scarlatina. The eruptive stage lasts from three to four days, and is usually free from severe general symptoms. It is occasionally followed by slight desquamation of the upper part of the thorax and thighs. During the height of the exanthema, there may be a rise of temperature, of two or three degrees, but it is only of short duration. As in measles, the mucous membrane of the throat is the seat of diffuse or dotted redness or yellowish miliary vesicles; the buccal mucous membrane, however, shows no Koplik spots. Most patients complain of sore throat during the acme of the disease, but not nearly as much as in scarlatina. The superficial glands, particularly the suboccipitals and those in the region of the angle of the jaw, the submaxillary, and less frequently those of the axilla, groin, etc., are enlarged and tender. In severe cases there is usually also moderate enlargement of the spleen.

The differential diagnosis between rubella and rubeola will be outlined on page 398. Attention will here be directed, however, to the frequent, nay, almost constant, occurrence of free perspiration in rötheln, a symptom, almost never met with in genuine measles. Where the rash is scarlatiniform, it may in the beginning be confounded with scarlet fever, but in the latter affection there are marked initial symptoms (vomiting!), high fever and pulse, and more severe throat manifestations.

Numerous so-called heat and stomach rashes greatly resemble German measles, and it is not always easy to tell them apart, particularly in the absence of an epidemic of rötheln. Under the circumstances it is safer to reserve the diagnosis for about twenty-four hours, and watch the results of a "cooling lotion" and a laxative.

For its differentiation from Dukes' disease, see p. 398.

Rubella is considered the mildest of all acute exanthematous infectious diseases, and, as a rule, terminates favorably within one week from the onset of the symptoms. But in view of the occasional occurrence of serious complications (severe angina, bronchopneumonia, suppurative adenitis, and even meningitis), it should always receive proper attention, especially in the way of rest in bed, light diet, cleansing of the nasopharynx, and good hygiene. (See also Treatment of measles p. 362.)

Diphtheria

Diphtheria is caused by a bacillus discovered by Klebs and Löffler in 1883. The bacillus is found in the secretions and excretions of the structures involved, and is transmitted usually through direct personal communication (kissing, sputum, etc.), but probably also through the agency of dishes, clothing, milk, etc., and through a third person, the so-called diphtheria carrier. The bacillus is very tenacious to life. It is said to remain in the throat of convalescents for several months or longer, and rooms previously occupied by diphtheria patients and left vacant for weeks frequently harbor infective diphtheria bacilli, having resisted disinfection and prolonged ventilation.

The diphtheria bacilli have a predilection for the lining of the naso-pharynx and larynx, especially of children from two to eight years of age. It is less common in infants under a year and very exceptional in the newborn.* Far more seldom they attack other parts of the body, *e. g.*, intestines, the eyes, the mouth, and the vulva. After imbedding themselves into the primarily affected structures, the bacilli multiply and secrete their toxins (albuminoses and organic acids), which enter the tissues and lymphatics and thence produce general infection. The diphtheria bacillus is often associated with strepto- and staphylococci and other bacteria.

Morbid Anatomy.—Diphtheria is characteristic for its formation of a fibrinous exudation produced by the entrance of the Klebs-Löffler's bacillus and other microorganisms into the superficial tissue layers. It is primarily a superficial destructive process which always ends in ulceration by separation of the tissues in the form of a true membrane. It differs from a croupous process only in the degree of severity of the inflammation. Gangrenous processes are sometimes associated with diphtheria, extensive putrid decompositions developing beneath the diphtheritic infiltration or upon the base of the diphtheritic ulcer. In healing, the entire necrotic process must first slough off, and the epithelium be replaced by scar tissue.

In all cases the neighboring lymphatic glands are swollen, hyperemic, intensely edematous and sometimes phlegmonous; and in severe

---

*Becker states that at the Jena maternity there were five cases of nasal diphtheria in newborn infants in 1918 and four in 1919. He warns that bacteriologic examination is indispensable for every case of coryza in a young infant and above all when the discharge from the nose shows traces of blood. In one case the nasal diphtheria entailed general sepsis with mixed infection and necrosis of the arm. The snoring breathing is the first symptom to attract attention, and then the thin, slightly purulent discharge running from one or both nostrils. It is often reddish or brownish, and erodes the upper lip. The membranes are generally far back in the nose, but can be easily removed. Becker ascribes the infection to carrier visitors as the most probable source. On this account it is now the rule not to give the child to its mother to nurse during "visiting hours," and no outsider is allowed in the infants' ward. In a recent compilation of thirty-eight cases the mortality was 31.6 per cent, mostly from complications. In another case the diphtheria settled in the cord.

cases there is usually an extension of the diphtheritic process from the pharynx to the nose, larynx, trachea and bronchi. Owing to its thick and dense epithelial covering the esophagus is very seldom involved. If the lungs become affected, the lesions usually consist of small lobular pneumonic foci, especially in the posterior lower portion of the lungs. Degenerative changes in the heart, spleen, liver, kidneys, intestines and cerebrospinal system are not uncommon evidences of the diphtheria toxin. The blood shows no definite changes.

Symptomatology.—The incubation period varies from two to ten days. As a rule, the onset is sudden with vomiting, headache, chills, fever, sore throat, and difficulty in swallowing. Not rarely however it is preceded by indefinite signs of ill health of a few days' duration, consisting of anorexia, lassitude, slight fever, irritation of the respiratory tract, etc. In such cases the active stage of the disease may insidiously follow upon the prodromic stage without any pronounced variation in the clinical manifestations, the throat symptoms often remaining latent until discovered by a routine examination or unmasked by grave correlative symptoms. This is especially apt to occur in infants. The importance of a routine examination of the throat of children in all kinds of complaints, therefore, is obvious.

The initial symptoms of the disease are not very characteristic, especially if the attack is mild. The uvula and tonsils are inflamed and somewhat enlarged. Careful inspection of the throat usually reveals upon the inner tonsillar or faucial surfaces a small, uneven, grayish-white, slightly elevated patch, or a few gray streaks or hemorrhagic specks. Within a few hours the deposit is found to have spread over both tonsils or also to the palatine arches and the posterior pharyngeal wall, giving the appearance of a greenish-white, sharply defined, firmly adherent membrane, which, if forcibly detached, leaves a raw, bleeding surface, and reforms very soon after. As the deposit assumes greater dimensions, the cervical and submaxillary glands, which at first are but slightly involved, become large and hard, assume the shape of large walnuts, and are very painful to the touch. Deglutition is difficult but not very painful—due to partial degeneration of the pharyngeal muscles and their nerves. The aforementioned constitutional symptoms continue.

The symptomatology thus far represents the first stage of a moderately severe attack of pharyngeal diphtheria. From now on three eventualities are possible: (1) The clinical picture may remain stationary; (2) the disease may spread to the nose from the pharynx; (3) the diphtheritic process may extend downward to the larynx.

PLATE VII

TONSILLAR DIPHTHERIA
(*Courtesy of Dr. John Zahorsky.*)

Since the introduction of antitoxin treatment of diphtheria the number of cases falling into the first category has enormously increased. With early treatment the disease is rapidly arrested, the membranes are cast off spontaneously, and the patient makes an uneventful recovery within from four to eight days. Less frequently the second or third possibility occurs. Either as a result of extreme virulence of the infection or of negligence or improper treatment, the nose or larynx or both becomes invaded. In nasal diphtheria (*rhinitis fibrinosa et membranacea*), in addition to the previously mentioned symptoms, nasal breathing is obstructed greatly. The child keeps the mouth widely open, snores, is very restless, speaks through the nose, is almost unable to swallow, has *fetor ex ore*, and coryza with seropurulent or hemorrhagic discharge. In laryngeal involvement (*diphtheritic croup*), symptoms of laryngeal stenosis predominate. The child's voice becomes husky, then hoarse, aphonic, and its breathing noisy, rough and wheezing, and as the disease advances, it is attacked by a barking, croupy cough, dyspnea, retraction of the lower portion of the sternum and the ribs with each inspiration, and cyanosis. The dyspnea often occurs in paroxysms, which greatly resemble those of spasmodic croup (*q. v.*), and grow worse from time to time. Unless the air passages are promptly freed from the obstruction by intubation (*q. v.*) or tracheotomy (*q. v.*), the patient passes into a state of stupor and finally succumbs to the effects of increase of carbonic acid and deficiency of oxygen in the lungs.

Both laryngeal and nasal diphtheria may develop primarily, and later become associated with pharyngeal diphtheria.

The course of the disease varies greatly with the location of the lesion, severity of the attack, and the period at which treatment is begun. Pharyngeal diphtheria usually pursues the most favorable course. Mild cases, as mentioned, may end in complete recovery in from four to eight days. In severer cases, the symptoms may increase in intensity up to the fifth or sixth day, and then begin to abate, and after a rapid or protracted course finally subside. The same holds true of nasal or laryngeal diphtheria, provided treatment is instituted early and no complications supervene. Unfortunately in the latter form of the disease complications are of quite frequent occurrence. Exhausted from the prostrating effects of the paroxysmal attacks of laryngeal stenosis, the child is unable to withstand the onslaught of the diphtheritic poison (sometimes also mixed diphtheritic and streptococcic infections). The deposit, originally limited to the upper portions of the larynx, rapidly extends *downward*, involving the trachea and bronchi—leading to croupous bronchitis and pneumonia, and, as a rule, to a fatal issue—and *upward*, exerting its destructive action upon the pharyngeal, oral and nasal

structures, often resulting in perforation of the palate, gangrenous sloughing of the uvula, etc. These cases of so-called *diphtheria gravissima s. maligna* sometimes develop very slowly and insidiously (*diphtheria larvata*) with symptoms of slight indisposition, slight rise of temperature, bronchial or gastrointestinal catarrh, and after a period of from a week to ten days are abruptly announced by true croup and the accompanying grave manifestations. Occasionally this form of the disease pursues a septic course right from the start, irrespective of the location and extent of the deposit. The virulent process is supposed to be due not only to the diphtheria toxin, but to the immediate entrance of the bacillus itself into the circulation. It is characterized by vomiting, prostration, puffiness and earthy pallor of the face; small, often irregular pulse; epistaxis; bleeding from the mouth, pharynx or into the skin. The urine is scanty, loaded with albumin; the temperature may be slightly raised or below normal. Within from three to five days the child dies, in a state of low muttering delirium, from gradual exhaustion, or earlier from cardiac paralysis. On postmorten examination, in addition to the diphtheritic lesions pathognomonic of all forms of the disease, the spleen is found enlarged, the kidneys, liver, and heart in a state of cloudy swelling—a group of pathologic findings ordinarily met with in severe infectious diseases—and, varying with the intensity and number of complications, divers lesions in other organs of the body (*e. g.*, lungs, brain and alimentary canal).

There is nothing definite about the number and severity of the complications in any given case. As already stated, mild cases may become severe and exhibit all sorts of complications and sequelæ and *vice versa*, cases with severe onset may under proper treatment remain free from either and end favorably in a comparatively short space of time. Kidney, heart, lungs and nerve diseases form the most frequent complications and sequelæ and in the majority of instances are the result of mixed infection. Transient albuminuria is often observed even in mild cases. It usually begins on the third or fourth day of the disease, sometimes earlier or later, and disappears with abatement of the other diphtheritic symptoms. Occasionally we find true *nephritis diphtheritica*, with large quantities of albumin and casts and more rarely also blood. The nephritis may also set in as a late sequel, during apparent convalescence, and remain more or less permanent. As a rule, however, the nephritis is of short duration, and rarely gives rise to local or general dropsy. By far more serious is the accompanying heart affection—so-called "heart paralysis" from involvement of the pneumogastric nerve. It is often manifested by sudden heart failure, and may set in either during the acme of the disease or any other time between then and as late as

from four to six weeks after; sometimes while the patient seems in very good health. It is apt to arise on the slightest exertion. The heart paralysis is not invariably sudden and fatal, however. Quite often it is preceded by heart weakness with symptoms of dilatation—interstitial myocardial degeneration—such as extreme pallor, feeble, rapid and irregular pulse, attacks of syncope, albuminuria, exhausting diarrhea, sometimes apathy, somnolence, sopor and death; or, less frequently, very slow convalescence, and gradual recovery, usually with remaining heart disease. Occasionally diphtheria is complicated by pericarditis or endocarditis. Bronchitis and pneumonia are especially prone to occur in laryngeal diphtheria, as a result of direct extension of the diphtheritic process to the trachea, bronchi, etc. (in intubated cases through the entrance of foreign bodies, particles of food, etc., into the air passages—"aspiration pneumonia") but also in other forms of the disease. The occurrence of pneumonia greatly mars the prognosis.

The most frequent sequel—occasionally also complication—of diphtheria is multiple neuritis, "diphtheritic paralysis," It is due to an intense degeneration of the peripheral nerves up to their roots. It follows in about one-tenth of all cases, probably mild and severe alike. It generally develops about the third or fourth week after the onset of diphtheria, sometimes earlier or later, and affects the muscles of the soft palate by preference, causing a nasal tone of voice, and regurgitation of fluids through the nose. In combined esophageal and laryngeal paralysis there is also great difficulty in deglutition, not rarely giving rise to "aspiration pneumonia," as a result of entrance of part of the food into the air passages. These disturbances usually disappear spontaneously or on suitable treatment, within from four to six weeks. The paralysis may extend to the eye muscles and cause strabismus, oculomotor paralysis, disturbance of accommodation and even total ophthalmoplegia. Less frequently the muscles of the trunk and extremities are implicated. (See Fig. 196.) The symptoms resulting are more or less identical with those observed in cases of multiple neuritis from other causes, and vary in intensity from simple motor weakness and ataxic gait to hemiplegia. In severe cases the tendon reflexes and faradic irritability are entirely lost, and the muscles undergo atrophy. Nevertheless, recovery is the rule in the majority of cases, except when complicated by paralysis of the respiratory muscles (diaphragm) and the aforementioned baleful sudden heart failure. As regards the hemiplegia, it is still uncertain, whether it is a genuine diphtheritic paralysis or caused by underlying alteration in the brain, such as cerebral hemorrhage, or cardiac thrombosis with embolism of the arteria fossæ Sylvii, since the hemiplegia not rarely begins with convulsions, loss

of consciousness, and is often associated with aphasia and facial paralysis. If the patient survives the attack, the hemiplegic symptoms usually subside within a few weeks, but weakness and contractures of the extremities may remain permanent.

Less common complications and sequelæ are arthritides, otitis, pleuritis, peritonitis, suppurative adenitis, diphtheritic affections of the stomach, diphtheritic ophthalmia, various rashes, etc.

From the foregoing discussion it can readily be appreciated that a positive prognosis is almost impossible. It should always be guarded, no matter how mild the case. The gravity of the epidemic, the sever-
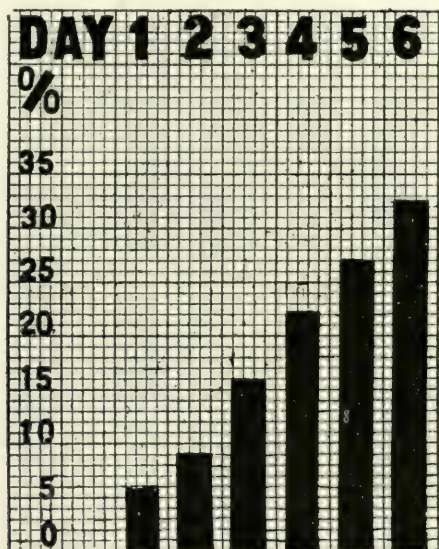
With Each Day's Delay in Giving Antitoxin, See How the Danger Increases!

CHART I

ity of the attack, the strength and age of the patient, the quality of the heart, the period at which antitoxin has been administered—all have an important bearing upon the outcome of the case. However, no case should be despaired of, no matter how grave. Antitoxin treatment often performs miracles, even in apparently hopeless cases.

Treatment.—With the advent of the serum treatment, diphtheria has ceased to be the dread of the community. The mortality of diphtheria which previously ranged between 50 and 75 per cent, has now dropped to about 5 per cent in pharyngeal and to 20 per cent in laryngeal diphtheria—the earlier the serum treatment is begun with the

lower the mortality. Indeed, by administering diphtheria antitoxin at the very inception of the disease we are often enabled to limit the latter to its *local* manifestations—almost free from constitutional symptoms. Furthermore, those coming in close contact with the diphtheria patient may by means of from 500 to 1,000 units of antitoxin be immunized against this affection for a period of from four to six weeks.

In a great many instances, especially in children's hospitals and asylums where large numbers of children are congregated, immunization may in many cases be dispensed with by employing Schick's toxin skin reaction (see p. 74), since it enables us to determine the susceptibility to or immunity against diphtheria. Moreover, as already emphasized on pp. 75, 76 permanent immunity may be effected by means of diphtheria toxin-antitoxin.

Immunization and isolation of the patient are the most potent prophylactic measures of diphtheria. As the nasopharynx forms the principal nidus for the development and spread of the diphtheria bacilli and their toxins, cleansing of the nasopharynx by means of mild antiseptics (instillation of Dobell's solution three or more times a day) will often aid in the prevention of infection. This prophylactic measure should be employed in conjunction with immunization by antitoxin, or without the latter, wherever there are contraindications to its use (*e. g.*, status lymphaticus, hemophilia) or objections on the part of the family. Heart disturbances being the most dangerous complication of diphtheria, the heart should receive very careful attention, even in the mildest form of the affection. It should be examined daily, especially as regards acute dilatation of the heart. The patient should be kept under observation for at least three weeks after abatement of the acute course of the disease, and in the event of any untoward symptoms arising, immediately be put to bed and treated in accordance with the directions presently to be outlined. Even with an apparently normal heart it is imperative to keep the child perfectly at rest in bed for at least ten days after disappearance of the local symptoms. As to the prevention of "aspiration pneumonia," the reader is referred to the chapter on "Intubation."

The active treatment of diphtheria can be summarized in a few words: counteract the diphtheria toxin; arrest the local lesion; and increase the power of resistance of the patient. When called upon to see a case of sore throat or laryngitis that is strongly suspicious of being diphtheritic in nature, we should immediately administer diphtheria antitoxin and lose no time in waiting for the results of a bacteriologic examination. The serum should be administered by deep hypodermic injections, a syringe somewhat larger than the ordinary hypodermic syr-

inge being preferably employed for this purpose. The lateral surface of
the abdomen or thorax or the outer surface of the thigh, where there is
an abundance of subcutaneous cellular tissue, is generally chosen for
the injections. Previous to the administration of the antitoxin the
skin should be carefully washed with alcohol or some disinfecting solu-
tion and the syringe carefully sterilized. Nowadays the serum is ob-
tainable in clean hermetically sealed syringes, rendering their steriliza-
tion unnecessary. Children under two years of age should receive from
3 to 5,000 units of antitoxin, and those over this age from 5 to 10,000
units. Equal or smaller doses may be given after about eight hours, if
no improvement is observed. The antitoxin injection is somtimes fol-
lowed (within from two days to two weeks) by an erythema or urticaria-
like eruption which usually disappears without any special treatment.
In malignant cases or in those seen late, double doses should be adminis-
tered at once and, if necessary, repeated, or the antitoxin may be ad-
ministered intravenously (after warming the antitoxin in its container
in hot water). The effect of the serum is very beneficial, nay, sometimes
magical. After a temporary rise, the fever often falls by crisis, the
pulse improves, the membranes loosen and disappear, and the whole as-
pect of the case sometimes changes completely for the better, within
from eighteen to twenty-four hours. However, notwithstanding all that
was said in favor of the antidiphtheritic serum, it is not always advisa-
ble to depend upon the serum alone.

As diphtheria is originally a local affection and the secretion and ab-
sorption of the metabolic products (toxins) occur from the local lesion,
the urgency of the immediate destruction of the bacilli at their point
of entrance is self-evident. This is best accomplished by the different
germicides and solvents, such as peroxide of hydrogen, strong solu-
tions of carbolic or salicylic acid, 20 per cent to 50 per cent solutions
of resorcin in alcohol, tincture iodine, argyrol or solargentum (20 per
cent), or the carbol-camphor solution referred to on p. 295. Milder so-
lutions of the same preparations should be used also for cleansing the
nose, even in the absence of any lesion there. The local treatment
should be repeated every two to four hours and continued until total
disappearance of the acute symptoms of diphtheria.

R	Glycerit. Papain.	℥ iv	
	Acid Carbolici	āā	15.0
	Pulv. Camphoræ	gr. x	0.65
	Alcoholis	℥ ii	8.0
	Glycerini	q. s. f ℥ ii	60.0

This is applied to the throat by means of a cotton swab every two
hours—changing the swab each time—diminishing the frequency of

applications with the abatement of the severity of the symptoms. Flushing of the throat with a warm solution of boric acid or bicarbonate of soda (2 drams to 1 quart of water) by means of an irrigator is very beneficial.

The third indication, to increase the power of resistance of the patient, should be met by an abundance of nutritious, easily digestible food, stimulants and hematinics. Feeding of the little patient is as difficult as it is important. As a rule, total anorexia prevails and it requires a great deal of patience and tact to induce the child to swallow a few mouthfuls of milk, beef juice, ice cream, fruit juices, etc. Still, much may be gained by administering the nourishment in small, frequently repeated quantities, and in small children, if need be, by rectal alimentation (peptonized milk). As a food and stimulant good wines and cognac are of inestimable value in diphtheria, especially in the septic variety. In malignant cases it should be given well diluted in large, frequently repeated doses (1 to 2 drams every two hours), preferably by mouth, and in urgent cases, cognac in smaller doses also hypodermically. It is advisable to employ mild stimulation from the earliest inception of the disease, and to continue it for weeks after in order to obviate—at least to a certain extent—sudden heart failure. A useful combination which acts both as stimulant and hematinic, is the following:

$\math1B$ Strychninæ Sulph.   gr.¼ | 0.01
Liq. Ferri et Ammonii acetatis ℥ ii | 60.0
M.
S.—One teaspoonful, diluted in sweetened water, every six hours.

Whenever the local as well as systemic effect of iron is desirable, the iron and myrrh mixture referred to on p. 391 answers the purpose admirably. Any untoward symptoms arising should be combated according to indications. In heart weakness, strychnine and digitalis should be pushed to full tolerance.

In laryngeal diphtheria without nasopharyngeal lesions, the local treatment outlined for the pharyngeal involvement may be dispensed with. Occasional cleansing of the nose and throat with a 5 to 10 per cent solution of argyrol, silvol, or solargentum or Dobell's solution, however, is useful as a preventive measure. It is of advantage also to have the patient inhale medicated vapors, such as the following:

$\mathit1B$ Acid. Carbolici   ℨ ss | 2.00
Eucalyptol   ℨ i | 4.00
Tr. Benzoini Compound q.s. ad f ℥ii | 60.00
M.
S.—One teaspoonful in a pint of hot water, for inhalation.

With early administration of antidiphtheritic serum the laryngeal stenosis rarely attains such severity as to demand relief by intubation (see p. 376) or tracheotomy (see p. 381). Mild paroxysmal attacks of dyspnea often yield to emesis (½ dram of wine of ipecacuanha, or 1/20 grain of apomorphine hydrochlorate), and a small dose of morphine (1/50 grain) and atropine (1/500 grain). But if these remedies fail, intubation or tracheotomy should be resorted to. It is always preferable to intubate (or tracheotomize) early rather than late. Whenever the dyspnea is steadily increasing in intensity and the temperature rises, this life-saving measure is indispensable, and procrastination is apt to prove fatal.

Differential Diagnosis

1. **Pharyngeal Diphtheria.**—(a) **Pseudomembrane.**—In pharyngeal diphtheria the pseudomembrane appears as a small, uneven, grayish-white, slightly elevated patch upon the *inner* or faucial surfaces of the throat. The deposit—which contains diphtheria bacilli—augments by quick spreading, reaching within a few hours the posterior wall of the pharynx, and, in severe cases, the Eustachian tubes, nares, and, more rarely, the conjunctiva. Anteriorly the pseudomembrane attacks the palatal arch and uvula. It may spread downward into the larynx or alimentary canal. The surrounding uncovered areas are grayish in color, due to overcrowding of leucocytes, nuclei, and mucus beneath. The tonsils, as a rule, are but slightly enlarged. The deposit, if removed, leaves a raw, bleeding surface and reforms rapidly.

In *follicular amygdalitis* the deposit begins as one or more white small pellicles upon the middle or anterior portion of the tonsil. The pellicles, at first distinctly isolated, gradually coalesce, forming elevated patches. They are limited to the tonsils, may easily be removed, and reform slowly. The tonsil, usually one, is moderately enlarged, sometimes previous to the appearance of the deposit.

In *parenchymatous amygdalitis* the tonsil is greatly enlarged, often displacing the uvula. It is bluish in color and doughy in consistency. The deposit, at first white, soon becomes yellowish, resembling the ''point'' of an abscess.

In *necrotic amygdalitis* or Vincent's angina the tonsils are moderately have a tendency to burst and leave superficial ulcers. This form of amygdalitis is at times accompanied by stomatitis. Otherwise it resembles follicular amygdalitis.

In *necrotic amygdalitis* or Vincent's angina the tonsils are moderately enlarged and the deposit lies deeply imbedded within the structure of the mucous membrane. The deposit, if removed, leaves behind a deep

ulcer—sometimes gangrenous—surrounded by a distinct red zone; it spreads, as a rule, from one tonsil to the other by way of the anterior pillars and palatal arch, frequently attacking also the uvula.

Vincent's angina and *septic sore throat* are best diagnosed by a culture from the tonsillar deposits.

(b) Submaxillary Glands.—The submaxillary glands in diphtheria are greatly involved. They are large and hard, assuming the shape of a large walnut, and can easily be seen protruding from the angle of the jaw. They are very painful to the touch.

In *follicular and herpetic amygdalitis* the glands are moderately enlarged, softer in consistence and less painful to the touch than in diphtheria.

In *parenchymatous amygdalitis* the glands are moderately enlarged and diffuse, the swelling often extending as high as the ear.

In *necrotic amygdalitis* the glands differ but slightly from those of diphtheria and cannot be relied upon as a differential point of diagnosis.

(c) Early Constitutional Symptoms.—Except the presence of albumin in diphtheritic urine, none of the early constitutional symptoms are characteristic of diphtheria. Indeed, they are frequently less pronounced in diphtheria than in any other throat affections, unless the former is complicated by streptococcic infection. The temperature in diphtheria, as a rule, is moderate, about 101° to 103° F., and continuous. The pulse is feeble and quick and soon gives signs of exhaustion. The face, as a rule, is pale. Swallowing is difficult, but not very painful, due to partial degeneration of the muscles of deglutition and their nerve supply. Albuminuria is invariably present from the earliest beginning of the disease and is of great significance in the differential diagnosis.

In the various forms of amygdalitis the temperature is quite high, especially toward evening, often reaching 105° F. The face is flushed. Deglutition is painful and difficult as a direct result of soreness and sensitiveness of the tonsils. Albuminuria is usually absent.

The diagnosis of *scarlatinal angina* is at best very difficult. It may be taken for granted that the primary amygdalitis of scarlet fever is scarlatinal in nature, and that the sore throat which sets in several days after is diphtheritic. It should be left, however, to the bacteriologic test to clear up the diagnosis.

2. Laryngeal Diphtheria.—Laryngeal diphtheria can only be mistaken for nondiphtheritic membranous laryngitis (see p. 310), and spasmodic laryngitis. In both of these affections, however, the Klebs-Loeffler bacillus is absent.

Intubation in Laryngeal Diphtheria

Before discussing the subject in question I deem it opportune again to recall the great services rendered by the master of intubation, the late Dr. Joseph O'Dwyer, of New York, who after numerous failures and discouragements finally succeeded in presenting to the world a priceless gift in the form of an intubation set, which has saved multitudes of children from gradual, agonizing death. Before this marvelous invention was fully accepted by the medical profession, Dr. O'Dwyer had been frequently humiliated by incompetent and possibly envious critics, rather than honored, remunerated, and decorated, by his state or country, or perpetuated in bronze or granite, as he surely would have been, had he been as successfully engaged in the acts of destruction, in the art of warfare, instead of in a deed of mercy.

My records of the past five years, during which time I have had the privilege of intubating sixty-eight children suffering from laryngeal diphtheria, show but one fatal issue. This favorable result was undoubtedly due to the facts, first, that the physicians in attendance had promptly administered ample doses of antitoxin to neutralize the diphtheritic toxin; and secondly, that the intubation was done early. It may be noted that all these children remained in their own homes, often in most undesirable surroundings, and without skilful nursing. Four of them lived out of town, requiring two or three hours' journey to reach. To emphasize the absolute feasibility and perfect safety with which intubation can be performed even under the most trying conditions, we may briefly relate the following case:

L. P., four years old, the son of Slavish parents in the poorest district of Perth Amboy, N. J., had been ill for three days before consulting Dr. S. Finding involvement of the nasopharynx and larynx, he immediately administered 10,000 units of antitoxin and prescribed other remedies ordinarily in use. During the night the child got very much worse and the laryngeal stenosis had assumed alarming intensity by the time we arrived there. Owing to considerable tumefaction and unusual depth of the larynx, intubation was somewhat difficult, but the boy obtained prompt relief with introduction of the tube. After giving 10,000 units of antitoxin and ordering absolute rest, we left the child, practically without any specific directions, under the care of the mother, who was entirely helpless and unable to understand our language. Five days later we returned for extubation, and, to our great amazement, we were met at the door by the little patient, tube still in the throat, but apparently perfectly happy. Extubation was comparatively easy, and the boy required no after-treatment whatever.

This case, among several similar ones, has fully convinced me that intubation can be performed without hesitancy even in the humblest of homes without any preparations or skilful after-treatment. This optimism is not shared by a goodly number of clinicians, one group of

whom, discarding intubation entirely as a dangerous operation and preferring tracheotomy, with the certain dangers and disadvantages of hemorrhage, secondary infection, tracheal fistula, stricture of the larynx, delayed convalescence from a slowly healing wound, and a disfiguring cicatrix; and another group of physicians, who, though recommending intubation in preference to tracheotomy, are nevertheless quite timid in accepting it as the operation of choice in all cases of laryngeal stenosis. They lay particular stress upon the risk of the tube causing ulceration of the larynx, or the danger of return or increase of the dyspnea, either by pushing false membrane before the tube or blocking it while in the larynx, and also of expulsion of the tube by coughing or otherwise. Now, we do not at all hesitate to say that those who claim intubation to be a dangerous operation never had the opportunity or inclination to learn the operation under the guidance of a competent instructor, nor have they given it a fair trial. In the many years of experience with intubation we have never met with the aforementioned difficulties, and believe that this is due, first, to the fact that with the early administration of antitoxin the virulent types of diphtheria of olden times are of very exceptional occurrence nowadays; and secondly, to the care and scrutiny in the selection of the cases and strict attention to the principles and technic as handed down to us by the late Dr. O'Dwyer.

To begin with, we must be absolutely positive that the cases in question actually require intubation. On several occasions we have been invited to intubate children who, instead of suffering from laryngeal stenosis, were in reality in the last stages of pulmonary edema, complicating pulmonary or cardiac disease. Recently I was called to intubate a fifteen months old infant supposedly dying from diphtheritic laryngeal stenosis. The baby did have tonsillar diphtheria, but no trace of laryngeal involvement, the noisy breathing having been due to intense dyspnea accompanying myocardial disease. I declined to intubate, and advised heart stimulants. The parents of the child, however, could not be reconciled to this view, seeing that the "baby was choking," hence insisted upon getting a throat specialist to intubate. This was done two hours later, with the result that the baby died during the operation.

The next point of importance before proceeding with intubation is to be certain that the instruments are in perfect working order.*

---

*A set of intubation instruments (O'Dwyer's) suitable for children up to the age of puberty consists of six tubes, an introducer, an extractor, a mouth gag, and a scale of sizes. O'Dwyer's latest tubes are made of hard rubber lined with gold-plated metal. Each tube is supplied with an obturator, one end of which screws on the introducer. The tube is selected according to the age of the patient—the smallest size for the first year, the second for the second year, the third for from two to four years, and the others, successively for children two years older. It should be remembered that the tube must fit the larynx and the latter not be made to fit the tube.

Particular attention should be paid to the construction and condition of the tube, more particularly that it be free from rough or sharp metal edges; otherwise, when during the act of swallowing the epiglottis and upper end of the tube are pushed posteriorly by the backward pressure of the base of the tongue, and the lower end of the tube is pressed forward, the gliding movement of the rough tube is very apt to injure the anterior wall of the trachea and thus to produce the ulceration of the larynx previously spoken of. We must also note that the obturator fits snugly, and that the tube selected corresponds to the size of the child's larynx.

After having ascertained these details to our entire satisfaction we may then proceed with the operation. The patient is placed upon a strong table, and, from shoulders down, wrapped tightly in a small
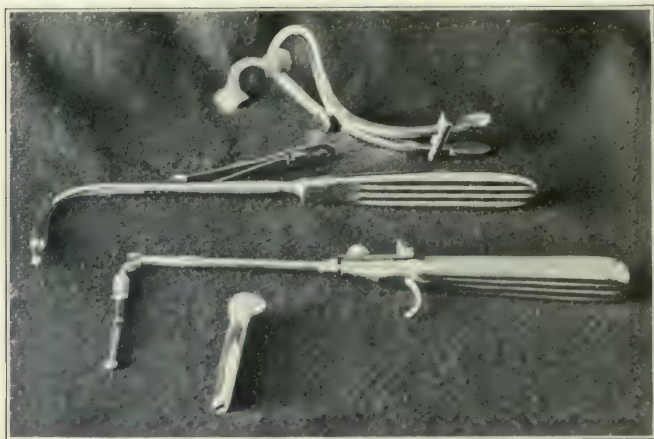
Fig. 88.—Instruments for intubation. (Dr. O'Dwyer's.)

sheet or blanket, fastened by several strong safety pins. An assistant standing at the head of the table inserts a mouth gag in the left angle of the child's mouth, well back between the teeth, and opens the gag as wide as possible without using undue force. The same assistant steadies the patient's head and holds the gag in place. The operator, standing to the right and in front of the patient, holds the introducer lightly between thumb and fingers of the right hand, with the thumb resting just behind the button that serves to detach the tube, and the index finger in front of the trigger underneath. The index finger of the left hand is now gently passed into the pharynx, down to the beginning of the esophagus, and by bringing the finger forward in the median line and raising and fixing the epiglottis, the tube (threaded

with silk to prevent it from slipping into the stomach in case it is wrongly put into the esophagus) is gently introduced along the left index finger into the larynx. The left index finger is then quickly put on the shoulder of the tube, and the introducer (with obturator) is withdrawn after pushing its upper button forward. After the tube has been securely pushed home the mouth gag is removed, but the silk thread is left in the tube for about ten minutes, until it has been ascertained that the dyspnea is relieved and no loose membrane is

Fig. 89.—Mode of feeding after intubation.

crowded down in the lower portion of the trachea. In removing the thread, the finger should be reinserted to hold the tube in place. As a rule, introduction is followed by an active spell of coughing, which generally expels mucus and bits of membrane that may have been lodged in the upper respiratory tract. Should we fail, however, to relieve the dyspnea, it is advisable to remove the tube immediately by pulling the thread, to induce emesis and expulsive coughing by tickling the child's palate and throat with spoon or finger, and then to reintroduce the tube, or, if the case be very urgent, to use a smaller tube

temporarily. There is never any danger in repeated intubation, or even failure to intubate, provided the operation is performed very gently—more particularly so as not to force a false passage—and the index finger is not allowed to rest upon the upper portion of the larynx too long, so as to obstruct the air passage.

The after treatment consists in keeping the patient quiet, preferably in a recumbent posture, application of an ice collar around the neck, and administration of antitoxin (if needed) and small doses of bromide, strychnine, and strophanthus. Feeding may be resumed a few hours after intubation: in babies, breast or cow's milk in small quantities, by means of a spoon; in older children, semisolid substances, such as custards, wine jelly, junket, soft-boiled egg, ice cream, and the like. Small pieces of ice may be given instead of water. It is often of advantage to feed the child with the head lower than the body (Fig. 89).

With the absolute subsidence of the dyspnea and temperature, which usually occurs in from three to seven days, we may proceed with extubation. It is always advisable to have another tube ready for immediate reintubation in case removal of the tube is followed by return of intense dyspnea. For extubation the patient is prepared in the same manner as for intubation. The extractor is guided along beside the left index finger in the same manner as the intubator and very gently inserted into the aperture of the tube. The engaging terminal blades of the extractor are opened by lightly pressing upon the upper arm of the extractor, and the latter is then promptly withdrawn from the throat. This maneuver is not always easy, but even repeated failure will do no harm, provided no force be employed. Occasionally one succeeds in removing the tube by "stripping" the larynx from below upward with one hand, at the same time grasping the head of the tube between the index and middle fingers of the other hand.

As a rule, these procedures end the operation. On rare occasions, however, there is an immediate return of the asphyxia. In this event, unless the dyspnea resumes extraordinary gravity, we may administer an emetic (apomorphine) or minute doses of morphine and atropine hypodermically, and spray the throat with a 1 or 2 per cent solution of cocaine until the spasmodic stenosis has been relieved.

I recall but two instances where I was obliged to reintubate three times, and one of them failed to show diphtheria bacilli in the throat after repeated laboratory examinations. In these cases, which are generally spoken of as "retained intubation tubes," we usually remedy the trouble by gradually introducing larger tubes (anointed with

vaseline) with each reintubation and by local attention to the nose and throat.

To counterpoise the admonition frequently given, "never to intubate patients who are extremely asphyxiated" (E. W. Goodall, Intern. Med. Ann., 1907), we may be permitted to relate the following instructive experience:

J. D., five and a half years old, had been coughing croupy for three or four days, and, as the parents were poor, was treated by them with the usual home remedies. In the middle of the night his condition became so alarming that they hurried for a neighboring physician. Finding that the boy was suffocating from diphtheritic laryngeal stenosis, the doctor promptly summoned me to perform intubation. As I entered the dingy and foul-smelling room I was greeted with, "It is too late, Doctor." Indeed, the boy was actually in the last stage of asphyxia, his face bluish black, his eyes protruding and suffused, his breathing suspended, and his entire body perfectly limp—apparently dead but for a barely audible fluttering of his heart. I remarked to my colleague that since we were not going to be paid for our visit anyhow, we might as well gain something from the additional practice in intubation. Thereupon I quickly inserted a tube in the boy's larynx, carried him to the front of an open window and injected $\frac{1}{20}$ grain of strychnine hypodermically, while Dr. F. proceeded with artificial respiration. There was shortly a marked change for the better and the child improved so rapidly that, after administering 10,000 units of antitoxin, we were able to leave him under the care of his mother within about an hour after our arrival. I extubated six days later, and the boy recovered fully without any further attention.

Tracheotomy

This operation is indicated where intubation fails to give relief, whenever the larynx is obstructed by foreign bodies, edema of the glottis, tumors (e. g., multiple laryngeal papillomas, or compression by tumors of neighboring structures) and cicatricial constriction of the larynx. Unless there be enlargement of the thyroid, the low operation is to be preferred, and, according to Donald Guthrie, may be performed without loss of blood, if the directions here given are followed:

The child is wrapped in a blanket or sheet to control its struggling and placed on the table. A pad of some sort is put under the shoulders, and the head is hung over the end of the table—steadied by an assistant. The operator stands at the right hand side of the child and, steadying the skin with the left hand, makes an incision in the midline of the neck from 1½ to 1¾ inches long. The skin and the superficial fascia are incised and the wound is held open by a pair of catspaw retractors which should not be more than an inch in breadth. When the deep cervical fascia is cut, the parallel branches of the anterior jugular veins are seen in the wound. The retractors are reset to pull these veins aside, and the sternohyoid and sternothyroid muscles are separated by blunt dissection.

If care can be exercised during this step of the operation, the muscles can usually be separated without injury to the thyroid ima beneath. The retractors are again reset, the left blade holding aside the skin, the fascia and the two muscles, and the right blade the skin, fascia, the muscles and the thyroid ima vein. This exposes the trachea to view. It is incised, the child's head is straightened, and the tracheotomy tube in-
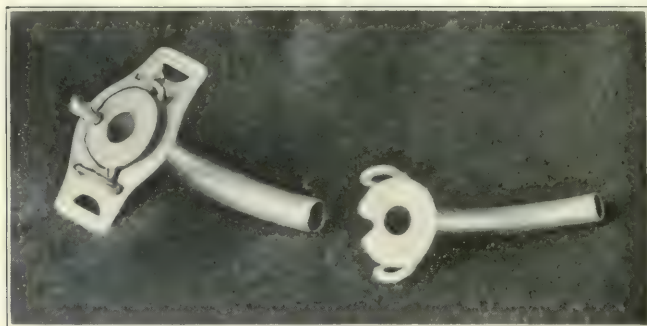
Fig. 90.—Tracheotomy tube.

serted. The tube should be removed from time to time in order to determine if the child can obtain a sufficient supply of air through the larynx. When this is achieved the tube is removed for good.

Scarlatina

(Scarlet Fever, Febris Rubra)

The more frequently one has occasion to observe and to treat scarlet fever, the more he appreciates the treacherous nature of the affection. Grave danger often lurks in the most benignly appearing attack, and dreadful surprises are not rarely encountered at a time when the patient is apparently at the threshold of recovery. It may be so mild in one child as to entirely escape observation, and yet may give rise to a most virulent type of the disease in another child. It is highly contagious and infectious in all its stages, the contagium (which is still unknown) being transmitted from person to person, through a third person, disease carrier, articles in use, toys, food (infected milk), and possibly also through the air. Children of from two to seven years are especially prone to contract the disease, but it has been observed even in the newborn of mothers suffering from scarlatina just before delivery, and also in adults. It prevails principally during the winter months. So-called surgical scarlatina is occasionally contracted after severe burns or surgical operations. As in other contagious and infec-

Seasonal Prevalence of Scarlet Fever
New York City, 1912 to 1916
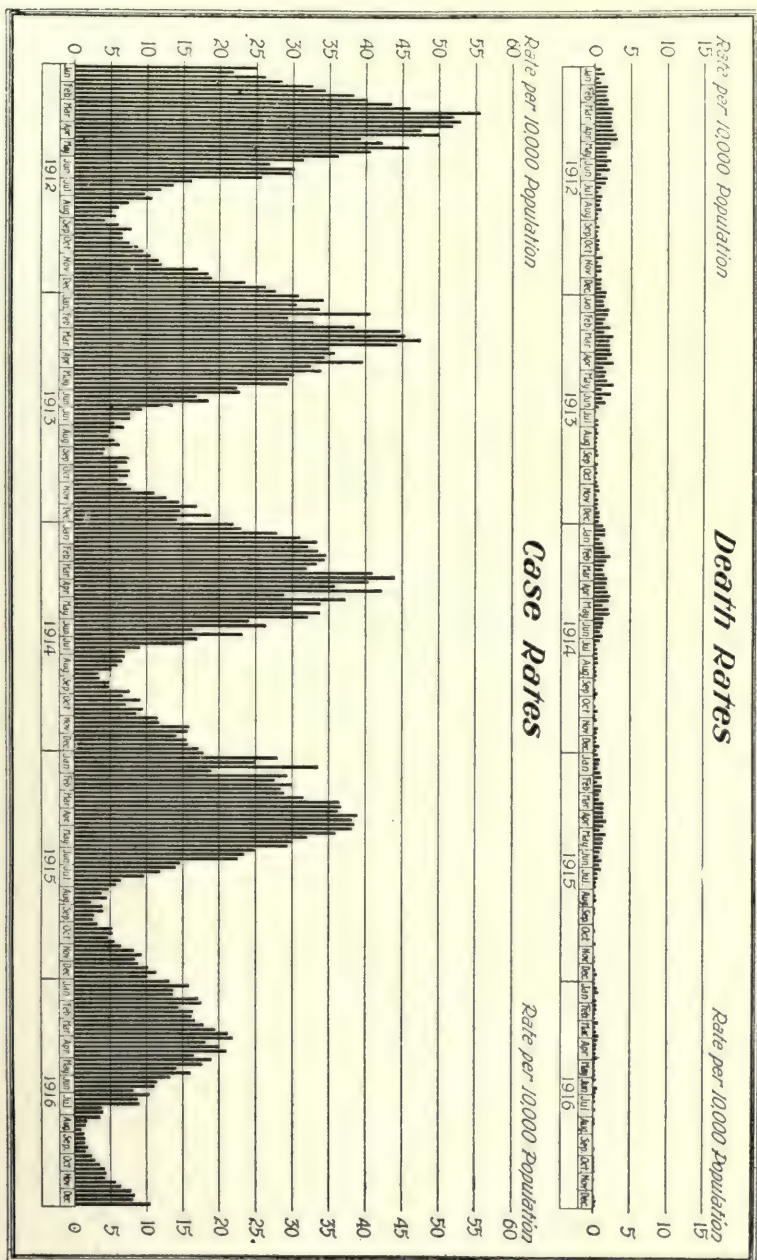
CHART II

tious diseases, some individuals possess an inherent or acquired tempo-
rary or permanent immunity against this disease. On the other hand,
some children are highly susceptible to scarlatina and may have two
or three attacks, sometimes even in the form of a relapse within from
two to six weeks after the first attack (*scarlatina recurrens*).

The incubation period of scarlet fever is ordinarily shorter than that
of any other exanthematous febrile disease. As a rule, it lasts only a
few days (varies from one day to one or two weeks), and rarely gives
rise to distinct symptoms of the approaching disease. On the contrary,
often in the midst of apparently good health, the patient vomits (usu-
ally repeatedly), complains of fatigue, slight sore throat, and chilliness;
and young nervous children are occasionally attacked by convulsions.
The temperature rises up to 103° or 104° F., or higher; the pulse is
greatly accelerated; the throat is deeply injected; the tonsils are some-
what enlarged and covered with a slight mucopurulent or hemorrhagic
deposit. Sometimes a transient, prodromal erythema is observed on dif-
ferent portions of the body. The aforementioned symptoms continue
for about twenty-four hours. By this time, or a few hours later, a bright
red rash becomes visible on the neck, chest and nates and the flexor
surfaces of the extremities. On close examination the eruption is found
to consist of very fine, rose-red to deep-red dots separated by minute,
pale areas of healthy skin. The scarlet points are not elevated above the
surface. The rash disappears on pressure, and when the finger nail or
a pencil is drawn across the reddened surface, a white line (*taches scarla-
tinale*) develops which remains *in situ* for a few seconds. This is due
to increased contractility of the superficial arterioles. Or if a tight band
is put around the upper arm we may shortly notice minute linear hem-
orrhages at the bend of the elbow (Rumpel-Leede sign). Gradually the
scarlatinal eruption spreads usually from above downward over the en-
tire body. It is least marked upon the face, and the circumoral ring—
a space extending from the alæ nasi to the chin—is nearly always free
from the exanthema. The affected skin is very itchy and often edema-
tous. With the advent of the eruption the temperature rises, the sub-
maxillary glands swell up, are hard and painful to the touch. Inspec-
tion of the throat in the majority of cases reveals a follicular deposit
upon the tonsils which shows a tendency to coalesce and to form necrotic
patches. The tongue is coated, very gray, and its edges and tip are
bright red. The papillæ fungiformes soon project through the coating
as red papules—"strawberry tongue." In accord with the height of the
temperature, the patient is more or less thirsty, restless, delirious, re-
fuses food, sometimes vomits; his urine is scanty, high colored, and usu-
ally contains a trace of albumin. The symptoms thus far related repre-
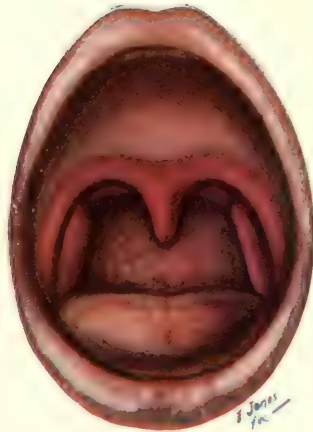
PLATE VIII

ANGINA SCARLATINOSA AND "STRAWBERRY TONGUE"

(*Courtesy of Dr. John Zahorsky.*)

sent the clinical picture of typical scarlatina during the first two or three days of the *eruptive stage*. As the disease advances the gray deposit on the tongue is cast off, the entire tongue is more or less swollen, red, often fissured, and covered with thickened papillæ. The deposit in the throat loses its tenacity, and sometimes falls off *en masse,* leaving behind raw, sometimes bleeding surfaces. The pulse and temperature (103 to 105° F.) continue quite high. Cases of considerable severity
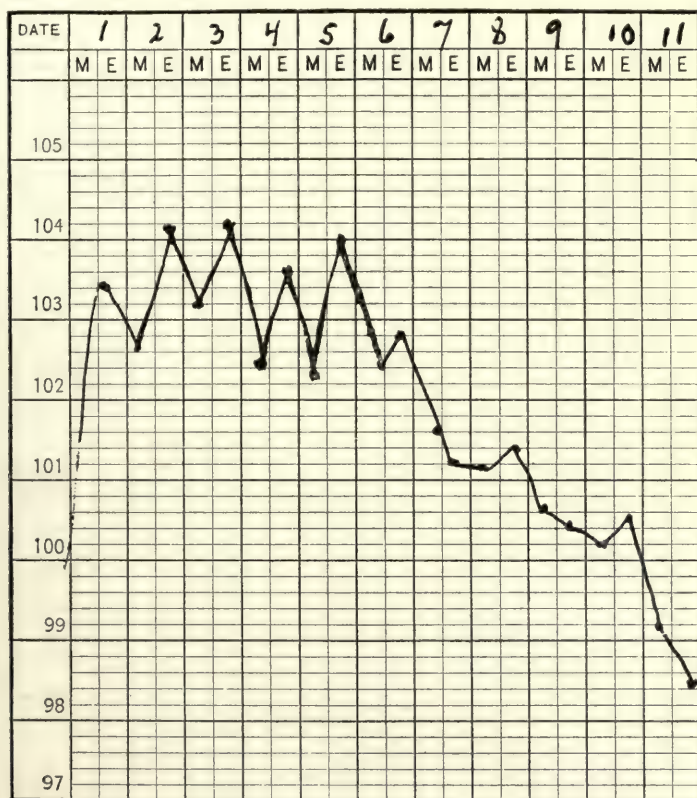
Fig. 91.—Fever curve of a case of scarlet fever.

present in addition marked debility; febrile, cardiac, systolic murmurs; slight enlargement of the liver and spleen and at times somnolence, delirium, with or without high temperature. On the other hand, mild cases by this time (fifth day) may be on the road to recovery, free from fever and rash, the patients being ready to be around and about.

The *stadium desquamativum* usually sets in four or five days after the appearance of the eruption, and depends somewhat upon the intensity of the exanthema, beginning earlier when the rash is pronounced. The peel-

ing may vary from fine branny scales to large patches of epidermis, the coarser scales being usually limited to the hands and feet. Occasionally the nails shed with the epidermis. The peeling may last from two weeks to as many months, or even longer. In uncomplicated cases desquamation is followed by decline of the symptoms and convalescence.

Complications are quite frequent, and their appearance is usually manifested by recrudescence of the temperature after defervescence. Scarlatinal angina—a necrotic inflammation of the throat—heads the list. It is caused by streptococcic infection and differs clinically from true diphtheria in that it almost never spreads to the larynx nor causes paralysis. Occasionally it is associated with true diphtheria.

The throat involvement may be grave right from the beginning of the scarlatina, but more frequently it develops between the third and fourth days, usually in the form of an aggravation of the previous condition. The glands at the angles of the jaws swell at times enormously, are very hard and tender. Inspection of the throat reveals a large yellow or gray exudate on the greatly enlarged tonsils, and often also on the posterior pharyngeal wall. Scarlatinal angina often extends also to the nose, giving rise to a fetid, brownish-yellow discharge, and occasionally to deeper destructive processes and even to necrosis of the nasal bones. Scarlatinal angina is a very malignant affection, and frequently leads to fatal termination as a result of gangrene of the throat, involvement of the neighboring blood vessels, purulent inflammation of the serous membranes (pleura, pericardium and meninges), extreme prostration, and general pyemia. In some epidemics one is able to distinguish two additional types of angina: 1. The "pestilential form," characterized by mucopurulent, foul masses in the throat and nose, spreading of the gangrenous process to the mouth and the mucous membrane of the lips and cheeks with consecutive hemorrhage, septicopyemic symptoms, increasing collapse, and fatal termination within about one week. 2. "Lentescent scarlatinal diphtheroid," which sets in about the sixth day of the disease with sudden rise of temperature, grave constitutional symptoms and intense swelling of the submaxillary glands. The local symptoms (which, by the way, are sometimes hidden!) in the nose and throat resemble those of true diphtheria, except that in scarlet fever there is a greater tendency to necrosis of the affected portions, and to perforation of the palate (as in syphilis). After stubborn persistence it quite frequently leads to fatal issue with symptoms of pyemia and asthenia. True diphtheria may be associated with any of the aforementioned forms of scarlatinal angina. An examination of the deposit for Klebs-Löffler bacillus,

therefore, is always opportune. Purulent otitis frequently arises as an immediate sequel of the nasopharyngeal involvement by extension of the inflammation through the Eustachian tube and tympanic cavity. It is manifested by the usual symptoms of otitis media: earache, restlessness, rise of temperature, congestion and bulging of the drum membrane, and, as a rule, rapid perforation of the drum by the pus. In a great many cases the otitis leaves no serious consequences behind; in some of them, however, especially in those in which the escape of pus is delayed, scarlatinal otitis may lead to very grave consequences, such as deafness (in very young children deaf-mutism) mastoiditis, meningitis, etc.

Another serious sequel of the throat affection is *angina Ludovici*, an inflammation of the submaxillary lymph glands and the surrounding cellular tissue of the neck, extending from the submental region up to the mastoid process of the temporal bone. The inflammatory infiltration sometimes extends to the larynx and produces edema glottidis, and, by gravitation, the pus may enter the mediastinum and neighboring structures (leading to purulent pleurisy or pericarditis). It not rarely ends fatally with symptoms of septicemia, embolism or thrombosis.

Among the earlier complications of scarlatina we may mention also pneumonia with or without pleurisy, rheumatism (myositis, synovitis) and endocarditis. All of these complications are probably of septic origin. The pneumonia presents nothing characteristic, may be lobular or lobar in type. It usually runs a shorter course than primary pneumonia. Scarlatinal rheumatism occurs in two forms: Simple myositis, *i. e.*, a localized muscular infiltration, with sensitiveness on pressure, and vague "wandering" pain; and scarlatinal synovitis or arthritis which is manifested by pain, swelling and redness of the joints, especially those of the fingers and toes, rise of temperature, and other constitutional symptoms. Sometimes several joints are affected by leaps. As a rule, scarlatinal rheumatism is benign in nature; occasionally, however, the joints may undergo suppuration, leading to general pyemia with fatal termination.

In association with scarlatinal rheumatism, but often also without this, endocarditis forms a relatively frequent complication and sequel scarlatina. Indeed, the majority of cases of valvular heart disease in children, except, of course, those complicating primary rheumatic fever, are traceable to scarlatina. The endocarditis may at first be latent, and escape detection, and again, may usher in with very grave symptoms, run the course of ulcerative endocarditis, giving rise to emboli and

metastases in the liver, spleen, and kidneys, and end in sudden death or permanent valvular heart disease.

The blood shows a pronounced leucocytosis, a marked increase of eosinophiles (up to 15 or 20 per cent of all white cells) and, in severe cases, streptococci.

The treacherous nature of scarlatina is most poignantly illustrated by the occurrence of nephritis as a complication. In the midst of apparently perfect health, at a time when the eruption has entirely subsided, either with or without any tangible cause (often after a slight error in the diet), the child is suddenly attacked by headache, dizziness, sometimes vomiting and convulsions, and examination of the urine reveals an interstitial inflammation of the kidneys. As the disease advances the symptoms enumerated under "nephritis" (q. v.) are rapidly and fully established. The complication usually occurs between the end of the second and third weeks. Hence the importance of daily examination of the urine in all cases of scarlatina, irrespective of the type or degree of severity of the disease. The duration of the nephritis varies greatly according to its severity, and the promptness with which it is discovered and treated. Ordinarily it lasts from two to four weeks and ends favorably, but relapses are not rare, and the nephritis may go on to chronic renal disease. In fact, scarlet fever, as a rule, forms the principal cause of chronic nephritis in children. Protracted scarlatinal nephritis often gives rise to hypertrophy of the left ventricle and occasionally also to dilatation of the heart with consecutive symptoms of ruptured compensation (recurrent anasarca, dyspnea, etc.). Genuine scarlatinal nephritis should not be confounded with the transient albuminuria not rarely observed during the first week of scarlatina, and which most probably is due to hyperpyrexia. As regards uremia, and its grave accompaniments, the reader is referred to "acute nephritis" (q. v.).

More rare complications are the following: stomatitis ulcerosa and aphthosa, noma, gangrene and diphtheria of the genitalia, orchitis, vaginitis, gangrene of the skin and of the tapering extremities; various nervous disorders, such as meningitis, hemiplegia, aphasia, tetany, and psychoses; conjunctivitis, iritis, keratitis, choroiditis, neuroretinitis, retinitis albuminurica and sudden amaurosis (in one of our cases total amaurosis lasted over a week).

Aside from the sequelæ previously spoken of, scarlatina may be productive also of chronic purpura, chronic cutaneous affections (furunculosis), chorea, paralyses, marasmus, tuberculosis, etc.

For the differential diagnosis see Table, p. 398.

The discussion of the subject in question thus far relates principally to cases of scarlatina of ordinary severity. In these cases the diagnosis is usually quite easy, and the prognosis, except in the presence of serious complications, relatively favorable. We shall now endeavor to emphasize some of the numerous atypical forms.

Occasionally scarlatina is associated with an atypical eruption. Instead of the fine scarlet rash there may be variously sized papules or wheals upon a reddened base; minute vesicles (*scarlatina miliares*) or pemphigus-like blebs. The exanthema sometimes evolves gradually, requiring several days instead of hours as is the case in typical scarlatina. The rash may appear localized with intervening larger portions of normal skin (*scarlatina variegata*). Finally, there may be genuine scarlatina, with typical angina, nephritis, and even slight desquamation, without any exanthema (*scarlatina sine exanthema*). The diagnosis in all such cases is extremely difficult, and sometimes impossible, unless at the same time typical scarlatina prevails in the immediate surroundings, and the other symptoms point strongly toward this disease.

The course of the attack also may present great variations. It may be so very mild and brief as to escape observation, or run a mild, but protracted course, and remain free from complications. In the latter group of cases the temperature may be low, or remittent, with evening remissions and morning exacerbations (*typus inversus*). Fever may be absent entirely even in severe cases. Sometimes the temperature is very high (*hyperpyretic scarlatina*) from the beginning, giving rise to delirium, convulsions, etc., but subsides again after a few days, leaving the patient apparently unharmed. At other times, very high temperature is characteristic of malignant scarlet fever.

Scarlatina maligna, gravissima s. fulminans, fortunately is not of very frequent occurrence. In the majority of instances the grave manifestations are in full bloom within the first twenty-four hours of the onset of the attack. The child is suddenly seized with vomiting, rigors, delirium or convulsions, the temperature rises to 106° F. or even higher. The pulse is weak, rapid and irregular. Sudden collapse, coma, eclampsia and death follow in rapid succession (often within twenty-four hours). In another group of cases the course is more protracted, and typhoid in character. The temperature is not as high as in the aforementioned class, but is marked by evening exacerbations; the tongue is dry, the lips and teeth are covered with sordes, the abdomen is very tympanitic, and the stools are watery. The submaxillary glands are enormously enlarged. There are also signs of blood dissolution, extensive hemorrhages from the nose, gums, and stomach, which greatly enhance the (fatal) exhaustion. The rash is usually of a vio-

let color and hemorrhagic spots are scattered over the surface of the body. This form of scarlet fever is often spoken of as "septic, hemorrhagic scarlatina."

Appreciating the unreliability of the initial manifestations, the uncertainty in the further symptomatology, the diversity of the course of scarlatina and its great tendency toward grave complications and sequelæ, it is prudent always to be very guarded in expressing an opinion as to the outcome of the disease, no matter how mild (or severe) the attack. The mortality varies in different epidemics, from 4 to 40 per cent, and depends partly upon the age (it is high in children under four and over ten years old) of the patient and principally upon the number and severity of the complications and sequelæ.

Treatment.—In view of the high mortality it is essential to institute prompt prophylactic measures from the very inception of an attack of scarlatina. Rest in bed is indispensable even in the mildest cases, and should be enforced for at least two weeks (much longer in severe cases) from the beginning of the illness. For about the same length of time the diet should be restricted, avoiding all such articles of food as are apt to upset the alimentary canal and to irritate the kidneys. In the active stage of the disease the diet should consist of milk only, and, as the symptoms abate, light cereals, and thin broths may be added; in older children also small quantities of toasted bread and butter, fish (boiled), chicken, soft-boiled eggs, and similar light food — all free from salt and spices. Easily digestible food should be continued for several weeks after subsidence of all traces of the disease. These procedures form the most potent means of prevention of renal and cardiac disease.

In view of the frequency of ear complications every effort should be made, firstly by cleanliness of the nose and throat, to prevent infection of the Eustachian tubes, and secondly, infection arising, promptly to make a free outlet to the accumulated discharge. (See Otitis, p. 303.)

As regards isolation, room ventilation, and disinfection, see p. 68.

It is quite difficult to formulate rules for the active treatment of the disease. Every case is a law unto itself. We have no specific to combat the affection. Overdosing—but also underdosing— with medicines is to be deprecated. Very mild cases do best if left alone, except as regards prophylaxis. The recent attempts to favorably influence the course of scarlatina by means of convalescent serum are still in the experimental stage.

The average case being usually of medium severity, an attempt will here be made to outline a mode of treatment which is best suited to meet ordinary indications. The patient should be put to bed in a

well-ventilated room (about 68° F.), the diet restricted to moderate quantities of water and a little milk—in the absence of vomiting. Since at the onset of the attack vomiting is usually very marked, no medication per mouth should be prescribed, except, perhaps, a few minute doses of calomel and bicarbonate of soda. To relieve high temperature and nervous irritation, we order a warm bath every three hours. The baths have also a very salutary effect upon the kidneys by enhancing the elimination of the scarlatinal poison through the skin. Warm packs may be given instead of the baths. As soon as the vomiting has ceased, we increase the quantity of nourishment and direct our chief attention to the throat. The latter is swabbed every two hours with from 5 to 30 per cent resorcin-alcohol solution or with the following:

℞	Acid. Carbolici	℥ ss	2.00
	Pulv. Camphoræ		
	Resorcini	āā gr. x	0.60
	Alcoholis	℥ ii	8.00
	Glycerini	q. s. f ℥ ii	60.00
	M.		

S.—Apply to the diseased parts by means of a cotton swab every two hours.

It is often very useful also to flush the throat several times daily with a warm boracic or bicarbonate of soda solution (1 dram to 1 quart of water).

The nose should be cleansed freely with Dobells' solution or similar antiseptic. Often it will prove beneficial to instill in the nose once daily a 5 to 10 per cent solution of the newer silver preparations. In some cases the nose is heavily blocked with a profuse foul discharge which greatly interferes with respiration; in this event relief may be afforded by introduction of soft rubber catheter tubing, reaching from the nares down to the posterior nasopharynx. If dysphagia and tonsillar swelling are marked, we prescribe moderate doses of sodium salicylate, or one of the newer salicylate preparations, and the following mixture:

℞	Tr. Ferri Chloridi		
	Tr. Myrrhæ		
	Potassii Chloratis	āā ℥ ss	2.00
	Glycerini	q. s. f ℥ ii	60.00
	M.		

S.—One teaspoonful every three hours, for child four years old.

With the aforementioned therapeutic measures we are ordinarily successful favorably to proceed with the case up to the fifth day,—

the time when "scarlatinal diphtheria" is prone to appear. As it is almost next to impossible to differentiate scarlatinal from diphtheritic angina without a bacteriologic examination, it is sound and safe practice to administer diphtheria antitoxin in all cases of severe angina, especially if the exacerbation of the symptoms occurs by the end of the first week of the disease. We usually inject 5,000 units of antitoxin at once and repeat the dose as indications arise. In malignant cases this can be combined with antistreptococcus serum or vaccine. The local and internal medicines should be continued, however, except bathing, which should be discontinued as soon as the temperature comes down to 100° F. The heart's action should be carefully watched, and any irregularity or debility detected, promptly treated by means of moderate doses of strychnine, digitalis or strophanthus. The latter two preparations are particularly useful in secondary involvement of the heart muscle. With the dietary and hygienic precautions taken one is seldom confronted by grave scarlatinal nephritis. Ordinarily, the symptoms are limited to slight albuminuria with occasional casts and blood cells, which readily disappear upon the administration of a few doses of calomel and alkaline diuretics and diaphoretics, or urotropin in 2 to 5 grain doses three times a day, high flushing of the bowels and a few hot baths. But, as already suggested, occasionally the uremic manifestations are extremely violent (delirium, convulsions, coma, etc.), resisting all sorts of medication, and growing worse from hour to hour. In these uremic conditions two therapeutic measures have proved to us of particular benefit: (1) Morphine and atropine hypodermically; (2) lumbar puncture. For a child four years old we may administer 1/20 grain morphine and 1/500 grain atropine, to be repeated once or twice within twenty-four hours. In very bad cases both of these measures should be employed simultaneously. Their effect is often magical.

Where the uremic symptoms are slight, bromide with or without chloral per mouth or per rectum suffice to relieve the nervous symptoms. As to the management of protracted cases of nephritis, see "Nephritis".

Simple transient scarlatinal myositis calls for no specific medication. On the other hand, arthritis demands prompt attention, since in the majority of instances it is a manifestation of sepsis and if left alone is apt to lead to general pyemia. The salicylates internally and ichthyol externally seem to influence it very favorably, and where these measures fail and pus forms we should resort to a free incision and drainage—but not too hastily. The same holds true for cervical or

submaxillary adenitis which, though assuming very large dimensions, does not always suppurate.

For suggestions as to the treatment of the remaining, less common complications of scarlatina, the reader is referred to the discussion of the respective diseases.

An extremely difficult problem confronts the attending physician when called upon to treat a case of malignant scarlet fever. Do what you will, the treatment is seldom of any avail. Early administration of polyvalent antistreptococcic and antidiphtheritic serum sometimes saves life, and should always be employed, regardless of bacteriologic findings in the nasopharyngeal discharges. The same holds good for lumbar puncture, if meningeal symptoms predominate. High temperatures failing to yield to hot baths should be reduced by cold (80° to 90° F.) packs or baths. The heart should be kept actively stimulated by strychnine, strophanthus, digitalis, caffeine, diuretin, and suprarenal extract, the latter especially in hemorrhagic complications.

During convalescence particular attention should be paid to the alimentary tract and skin. The bowels should be looked after, and stuffing the child with sweets, heavy meats, and alcoholic "tonics" strictly forbidden. The patient should be warmly clad and wear flannel or silk next to the skin. Exposure to sudden atmospheric changes should be avoided.

To facilitate desquamation, the child should be given a hot soap bath every two or three days followed by oil inunction to prevent free distribution of the scales. The following combination is quite serviceable, and may be employed also in the eruptive stage of the disease to relieve itching and burning of the skin:—

℞	Thymolis		
	Acid. Carbolici	āā gr. x	0.65
	Alcoholis	℥ ii	8.00
	Glycerini	q. s. f ℥ ii	60.00
	M.		

S.—For external use, p. r. n.

When desquamation is completed and there is otherwise no contraindication, the patient may be allowed out of doors. Cod liver oil with the syrup of the iodide of iron and a sojourn at the seashore have proved very helpful to rapid recovery.

The patient is "contagious" for at least six weeks from the onset of the disease, and hence should not be permitted to mix with other children for that length of time, or longer, if desquamation continues, or discharges from the nose, throat, vagina, etc., are present.

The Fourth Disease*

(Dukes' Disease)

The existence of this affection is still awaiting authoritative confirmation. Some authorities maintain that it is merely a mild form of measles or scarlet fever. It begins after an incubation period of from six to fourteen days or longer with very mild febrile symptoms and an efflorescence on the face, including the circumoral ring. The next day the rash spreads, grouped in a sort of lacework arrangement, to the extremities and trunk. The course of the affection is conspicuous by absence of any severe symptoms and usually terminates favorably in from five to eight days, without any specific medication. Desquamation is copious and may last several weeks. Dukes' disease does not confer immunity against other exanthemata.

Varicella

(Chickenpox)

The identity of the causal microorganism of varicella is still unknown. It is absolutely proved, however, that it has nothing in common with the infectious agent of smallpox,—hence an attack of chickenpox confers no immunity against the former affection. The disease is communicable from person to person, through an intermediate person, through fomites, and the air. Children of from two to ten years of age are especially prone to contract the disease, but it is not rarely observed also in very young infants, and in children over ten, and even adults are not entirely exempt from it. One attack does not confer absolute immunity against another one.

The incubation period lasts about two weeks, the last few days showing slight prodromata. Occasionally the symptoms of invasion are moderately severe. There may be vomiting, angina, conjunctivitis, transient erythema, considerable rise of temperature preceded by chills, and in small children, convulsions. The eruption, which appears usually in small or large crops without any characteristic grouping simultaneously upon several portions of the entire body (also on the scalp and the mucous membrane of the mouth and throat) is fully established within twenty-four hours. At first the eruption appears in the form of slightly elevated rose-red spots, which disappear on stretching the skin. Within a few hours the center of the spot turns vesicular, filled with a clear fluid. The spots attain the size of a lentil or pea, but they may be larger, pemphigoid, and more rarely umbilicated. On the third day the vesicles usually collapse and desiccate, and

---

*Termed so, being additional to the three known diseases: Scarlatina, Rubella and Rubeola. It was first described by Dukes in 1900.

become covered by brownish-black crusts. The latter usually fall off on the fifth or sixth day, leaving slight red spots which soon disappear. Repeated recurrences of new crops of the eruption in different stages of development (papules, vesicles, pustules and crusts), sometimes as late as ten to twelve days after the onset, are not rare and often serve of signal value in the differentiation of varicella from variola, in which latter disease the eruption remains uniform and stationary until the final stage of the disease. Occasionally the vesicular content is turbid or purulent (usually as a result of infection by scratching), and when the pustules heal leave behind scars resembling "smallpox pits." Sometimes the vesicles burst early and give rise to erosions and ulcerations which, if occurring in the larynx, may be productive of attacks of dyspnea and, exceptionally, fatal laryngospasm. More frequently we meet, usually as a result of infection, with multiple ulcerative and gangrenous processes of the skin —*varicella gangrenosa*—in which the vesicles terminate in deep, foul-smelling ulcers, and extensive gangrene of the skin. This form of chickenpox is most common in delicate, ill-nourished children and is apt to prove fatal. Complications and sequelæ in the form of nephritis— *nephritis varicellosa,* pneumonia, pleuritis, pemphigus—*varicella bullosa,* multiple abscesses, pyemic processes (due to staphylococcic or streptococcic infection), icterus catarrhalis, dysentery, polioencephalitic manifestations, marasmus and even tuberculosis are on record, but they are rather of unusual occurrence. Finally varicella is occasionally associated with other exanthemas (*e. g.,* measles, scarlet fever). Very recently several clinicians have called attention to a curious relationship between varicella and herpes zoster and are inclined to the belief that the latter is an atypical manifestation of the chickenpox virus. This view is awaiting further confirmation.

Treatment.—As a rule, varicella pursues a benign and brief course, free from high temperature, and any other constitutional symptoms and rarely calls for any therapeutic measures. Rest in bed, careful diet, and local cooling lotions (2 per cent of thymol in albolene) or ointments (zinc oxide with 1 per cent salicylic acid and thymol and phenol) to relieve itching usually suffice in ordinary cases. Cleanliness of the mouth and throat is important, as well as attention to the urine. For differential diagnosis, see Table, p. 398.

Variola Vera. Varioloid

(SMALLPOX)

The history of smallpox is that of death and destruction. It is estimated that, before Jenner's discovery of prophylactic vaccination,

one tenth of all the children died of smallpox. On the other hand, with vaccination and revaccination rendered obligatory in most of the civilized countries, the occurrence of variola in a child is almost unheard of. If it ever does occur in successfully vaccinated children, the disease is usually mild, modified in form—*varioloid*.

Smallpox is an acute, highly contagious and infectious, endemic and epidemic disease, characterized principally by an eruption that passes through the stages of papule, vesicle, pustule and scab, the development of the pustule being accompanied by a secondary fever.

The nature of the smallpox producing poison is still unknown. It is undoubtedly a microorganism that exists in the eruption and probably also in the blood. Garnieri has described minute corpuscles which are regularly found in the cells of variola and vaccinia pustules, but while they may serve as a characteristic differential point from varicella and other pustular eruptions, they seem to be of no etiologic importance. The disease is most communicable during the pustular and desquamative stages—at which time mere entering the sickroom is said to infect one not protected by vaccination.

After an incubation period of from nine to fifteen days, which as a rule, is free from any significant signs of illness, the patient is suddenly seized by a violent chill, fever, severe pain in the back, convulsions, delirium, prostration, and sometimes collapse and death— long before the appearance of the eruption. An initial exanthema may appear at this time, in the form of an erythema or hemorrhagic spots, upon the trunk and extremities, more particularly on the anterior surface of the thigh (the so-called Simon's triangle). This mode of onset and termination is quite common in variola vera, affecting children under three years of age. Some cases survive until the appearance of a papular exanthema upon the buccal and pharyngeal mucous membranes, and then usually die from exhaustion; others again—usually older than three years—succumb to the attack in the suppurative stage, or, rather rarely, recover after a painful and tedious convalescence.

It is customary to distinguish three types of variola vera: Discrete, confluent, and malignant (hemorrhagic).

Discrete Form.—After the violent onset, the eruption, consisting of red, coarse spots, appears during the *third day*: first on the forehead and lips, then on the head, trunk and arms, and last on the legs. Pressing the hand over the eruption, the latter imparts the sensation of velvet. The constitutional symptoms then abate, and the patient feels quite comfortable. On the fifth day of the disease the spots develop into papules; on the *sixth* into vesicles which soon become umbilicated. On the *eighth* day the vesicles are transformed into pustules which

emit a characteristic odor and on the *ninth* day they become entirely purulent and surrounded by a broad red band—the halo or areola. The face becomes swollen and the features are distorted. On the *eleventh* day it is usually found that pus oozes from the pustules which on drying forms the scab or crust. The latter falls off sometime between the seventeenth to twenty-first days, leaving a red, glistening depression or pit which soon changes into a white cicatrix. With maturity of the pustules eighth or ninth day) the symptoms observed at the onset return—*secondary fever*. This fever of suppuration is the most critical period of the disease. In favorable cases the secondary fever abates after a few days and convalescence follows. The stage of suppuration is very prone to be complicated by severe inflammation of the larynx, bronchi, lungs, and serous membranes. As further complications or sequelæ we may mention stomatitis, noma, involvement of the eyes (phthisis bulbi), otitis media, dysentery and nephritis.

Confluent Form.—This form is characterized by extreme violence of the constitutional symptoms and by the confluence of the eruption on certain portions of the body, such as the thigh and lower portion of the abdomen and the neck.

Malignant or Hemorrhagic Form.—This type of smallpox is characterized by malignancy and irregularity of the symptoms, and coexistence of hemorrhages and petechiæ. In this form are included the so-called black smallpox (*variola hemorrhagica pustulosa*) which usually leads to fatal issue in the suppurative stage, and the fulminant type of smallpox (*purpura variolosa*) which ends fatally within from three to four days.

In contrast to variola vera with its dreadful consequences stands *variola modificata or varioloid*. The latter form of smallpox is usually observed in children rendered partially immune by previous vaccination or an attack of smallpox. Its course is shorter and milder than that of the other forms, the eruption is slight and devoid of suppuration,—hence its freedom from secondary fever and severe complications and sequelæ. The mortality in varioloid varies between 8 per cent and 10 per cent in infants and about 5 per cent in older children.

Smallpox may be confounded, in the initial stage, with meningitis and, in the eruptive stage, with varicella and morbilli, especially morbilli hemorrhagici (*q. v.*). Meningitis can readily be eliminated after a day or two. The differential signs between smallpox and the other exanthemata are outlined on p. 398.

Treatment.—If the patient with smallpox is seen early, vaccination should be performed at once; it may modify the attack.* As a prophy-

---

*In mild, doubtful cases vaccination may serve as a valuable aid in the diagnosis, for if successful, it would at once exclude the presence of smallpox.

DIFFERENTIAL CHART OF EXANTHEMATOUS DISEASES

	Rubeola	Rubella	Scarlatina	The Fourth or Dukes' Disease	Varicella	Variola
Incubation period (number of days).	9-15	10-21	3 to 10	6-14	12-14	9-15
Principal symptoms at onset.	Catarrh of naso-pharynx and eyes; Koplik's spots	Catarrh of nose and throat; rose spots on soft palate.	Vomiting; angina; hyperpyrexia; very rapid pulse.	Indisposition.	Slight febrile symptoms, sore throat	Headache, backache, chills; convulsions.
Time of appearance and character of eruption.	Fourth day: elevated red spots on face, spreading over entire body.	Second day: pale red maculæ on face; irregularly distributed on body	Second day; bright red fine scarlet rash on neck, chest and face (except around mouth).	First day: efflorescence on face; next day on extremities and trunk.	First day: crops of thin papules soon changing into vesicles which begin to dry on third day.	Third day: coarse papules on forehead and lips; spreading downward; changing into umbilicated vesicles, pustules and scabs.
Conspicuous symptoms during course	Moderate fever; tracheobronchitis.	Slight fever; glandular swelling especially on back of neck; sore throat.	Severe diffuse, angina, "strawberry tongue; desquamation	None	Moderate fever; sore throat; severe itching.	Disappearance of fever on third day; reappearance of "Secondary fever", on ninth day.
Principal complications	Pulmonary and ear disease.	The Same.	Diphtheria; otitis myositis; renal disease.	None	Skin infections	Respiratory disease; skin infections.

lactic measure it is also advisable to vaccinate all those who come and are apt to come in contact with the patient. Isolation, disinfection and preparation of the sick-chamber (the room should be kept dark by a deep-red shade) should be carefully carried out, in the manner prescribed on p. 69. The child should be confined to bed, and kept on a light but nutritious diet, and liberal supply of stimulants (wine, cognac). Special attention should be paid to disinfection of the mouth and nasopharynx (mild solution of potassium permanganate, or chlorate, peroxide of hydrogen). In high temperature and severe nervous phenomena prolonged warm baths or cool packs act favorably. To prevent itching and extensive pitting we may apply 5 per cent to 10 per cent of ichthyol in equal parts of zinc and sulphur ointments, covered by some unctuous material to exclude the air. It is sometimes necessary to tie the patient's hands to prevent scratching; and to administer hypnotics and anodynes for the relief of restlessness and pain. The child should be quarantined for about six weeks.

℞	Antipyrinæ	gr. xxiv	1.60
	Tr. Cinchonæ Comp.	ʒ iii	12.00
	Syr. Aurantii	℥ i	30.00
	Aq. Aurantii	q. s. ad f ℥ ii	60.00
	M.		

S.—One teaspoonful every four to six hours, for a child four years old.

℞	Mentholis	gr. v	0.30
	Bismuthi Subgallatis	gr. x	0.65
	Zinci Stearatis	℥ ii	60.00
	M.		

S.—Dusting powder to enhance desiccation of the eruption and to relieve itching.

Typhus Abdominalis

(TYPHOID, ENTERIC FEVER)

Typhoid fever is an endemic, epidemic, and sporadic infectious disease due to the bacillus typhosus of Eberth. It is characterized by a continuous, typical fever, gastrointestinal catarrh, and a roseolar eruption. With the recent advances in bacteriologic diagnosis we are now certain that typhoid occurs almost as frequently in children (even fetal typhoid is on record!) as in adults, but owing to the mildness of the clinical picture it is frequently overlooked. The younger the child the greater the deviation of the symptomatology from the usual course. Thus, the onset is either more protracted (with symptoms of subacute gastroenteritis) than in the adult or very sudden with chills

and high fever. In the newborn the symptoms may resemble those
of sepsis. In older children the initial stage (*pyrogenetic* stage, first
week) resembles that of adults and is marked by epistaxis, frontal head-
ache, anorexia, furred tongue (later dry and brown), restless sleep, and
gradual rise of temperature. The action of the bowels is not character-
istic, and constipation may alternate with diarrhea (sometimes bloody).
The fever reaches its height with the approach of the second week
(*fastigium*), and varies in mild cases between 101° and 103° F. and in
severe cases between 104° and 106° F., with morning remissions and
evening exacerbations ("step curve"). Occasionally the typus inver-
sus is observed, and not rarely the temperature is remarkably low
throughout the entire course of the disease. The pulse is sometimes
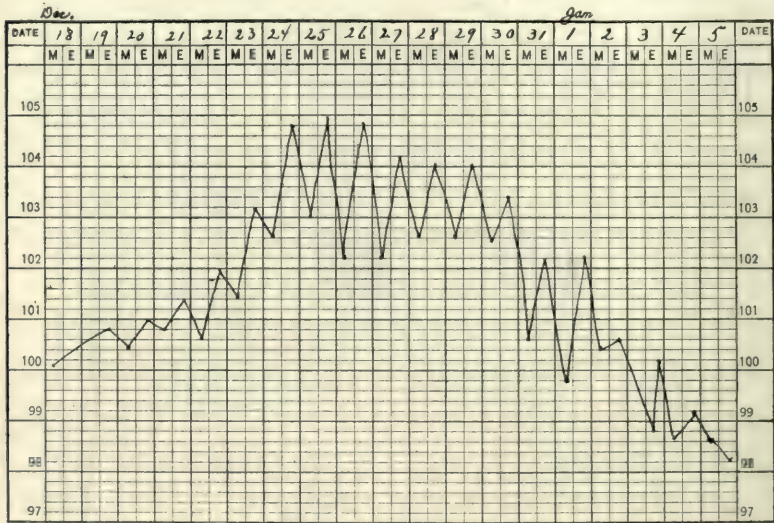
Fig. 92.—Fever curve of typhoid fever in child four years old.

very frequent (160 to 180) but rarely dicrotic. The urine responds to
the diazo reaction, and contains traces of albumin. During this stage,
the second week, the spleen is palpable, but not as distinctly as in adults.
The roseolar eruption which usually appears about the eighth day on the
abdomen, chest, back and limbs, is rather scanty and not rarely entirely
absent. The typical eruption consists of small, elevated, rose-colored
spots which momentarily disappear on pressure. They evolve in suc-
cessive crops, each crop lasting about three days, and subside entirely
after about ten days. Corresponding to the comparative mildness of the
intestinal lesions, tympanites, iliac tenderness and gurgling are rarely
marked. The same is true of the abdominal pain. If it is pronounced

we should look for a complicating cholecystitis, appendicitis, peritonitis or intestinal perforation. This last complication is most apt to occur in the third week of the disease, and sets in either insidiously or abruptly, in the latter event with a sharp fall and abrupt rise of temperature (often preceded by a chill or vomiting), meteorism and abdominal rigidity, and subsequent appearance of fluid in the peritoneal cavity. This process is usually accompanied by a more or less marked leucocytosis. During the acme of the fever there are more or less marked nervous phenomena. Some patients are drowsy and apathetic; some are restless, shriek, and rave; some suffer from defective hearing, hyperesthesia, insomnia, or semistupor, and, finally, others may be dull during the height of the fever but otherwise may be playful during the entire course of the disease. Children almost never present the status typhosus. As a rule, the blood gives a positive Widal reaction (*q. v.*).

With the beginning of the third week (*defervescent stage*) there is a decided improvement in the general symptoms. The tongue begins to clear at the edges, the appetite returns (is often voracious), the temperature declines, as a rule, by lysis, and the grave nervous symptoms gradually abate. The temperature sometimes drops suddenly and remains normal or even subnormal. In severe cases, however, the fever may continue (*ambiguous stage*) and with it all the other symptoms. Indeed, in older children the intestinal manifestations may become more pronounced, and hemorrhage from the bowels, perforation and peritonitis may supervene. The usual bronchial catarrh may extend to the bronchioles and pulmonary tissue and lead to diffuse bronchopneumonia. Furthermore, improvement and recovery may be greatly delayed or entirely arrested by relapses, which are not uncommon between the third and fifth weeks, or by the following complications and sequelæ: inflammation of the mucous membrane of the mouth (occasionally noma!), nasopharynx, and larynx; parotitis, otitis, cutaneous abscesses, periostitis, perispondylitis (typhoid spine); pericarditis, endocarditis, purulent arthritis, pyemia, thrombosis and embolism; paralyses (usually neuritis), chorea, aphasia (lasts about a week), dementia, maniacal and melancholy states. The mental sequelæ usually consist of merely temporary irritability, hypersensitiveness, disposition to cry, capriciousness and surliness. On the other hand, cases of permanent mental aberration are on record. Typhoid fever is sometimes associated with pertussis, morbilli, scarlatina and diphtheria, and in cases with a predisposition it is apt to be followed by pulmonary tuberculosis. Occasionally, typhoid is followed by a posttyphoidal desquamation of the skin, and during and after an attack there is frequently a marked longitudinal growth of the bones,

especially of the tubular bones of the lower extremities. As a result of it, the skin over these bones is sometimes transversely torn, the tears being indicated at first by red lines, and later by white scars.

The aforementioned grave complications and sequelæ, are very rarely observed in children. As a rule, the prognosis except in very young infants, is favorable (5 to 10 per cent mortality), and, even after severe attacks, convalescence is comparatively rapid and uneventful. In young children the course of the disease is usually very brief, between twelve and fifteen days; in older ones it is nearly the same as in adults.

The morbid anatomic condition in the intestines is much milder than in adults; ulcers are rare, and, if present, are small, superficial and isolated, hence they heal without leaving behind any cicatrices in the intestines or any tendency to cicatricial contraction.

In view of these marked deviations from the usual clinical picture, the diagnosis of sporadic cases of typhoid fever often presents great difficulties. It is apt to be mistaken for simple *gastroenteritis*—febrile stage of shorter duration, spleen, in uncomplicated cases, not enlarged, diazo reaction and Widal's blood test negative; *influenza* with pronounced intestinal symptoms—febrile ''step'' curve absent, nervous phenomena less pronounced, catarrhal symptoms more marked, Widal's test negative, *pneumonia*—more sudden onset, more positive pulmonary physical signs, Widal's reaction negative, diplococcus pneumoniæ in the expectoration, neutrophilic leucocytosis; *acute miliary tuberculosis*—irregular temperature with sweats, hectic flush, often tuberculous sputum, more protracted course, Widal's reaction negative; *tuberculous meningitis*—lower temperature; slow, irregular pulse and respiration; troughshaped abdomen; *malaria*—usually intermittent or recurrent fever, malaria plasmodium in the blood, influenced by quinine; *septic endocarditis* —pronounced heart symptoms, chills with septic temperature, absence of Widal's reaction; *tick or Rocky Mountain spotted fever*—endemic of this region, characterized by a continuous, moderately high fever, severe muscular and arthritic pains, profuse petechial or purpural skin eruption appearing first on the ankles, wrists and forehead. Widal reaction is negative; *typhus, spotted fever*—general malaise, irregular pain throughout body, continuous fever, ending by crisis on the fourteenth day. Macular, petechial rash usually on the third to sixth day upon body and extremities. Weil-Felix's reaction is positive. Widal's reaction is negative. Occasionally typhoid begins with pain in the occiput, neck and back, opisthotonos, and other grave nervous phenomena, presenting the clinical picture of *acute meningitis*. The diagnosis in such cases is often almost impossible in the first few days of the disease. In doubtful cases the bacteriologic examination of the cerebrospinal fluid

for the diplococcus intracellularis, and of the stools and urine for the bacillus typhosus often proves decisive.

Treatment.—As the contagium of typhoid fever resides principally in the gastrointestinal contents, it is imperative to disinfect the stools and vomitus thoroughly, as well as the linen and other articles in use that have been soiled by the discharges. Furthermore, by taking the precaution of boiling the drinking water or milk, excluding mosquitoes and flies from the sick-room, and by avoiding dissemination of the source of infection through soiled bath tubs, hands, etc., the disease may be limited to a single patient notwithstanding the intercommunication between the patient and other members of the family. Strict isolation, therefore, is not essential. Prophylactic immunization!

Typhoid fever is a self-limited disease and not controllable by any specific measures. The treatment, therefore, should be symptomatic, principally hygienic and dietetic. Cleanliness of the mouth and nasopharynx, cool sponging of the body, with water or alcohol or vinegar, or if the temperature is high, cool packs or full baths, at a temperature of from 80° to 90° F., and an ice bag to the head, usually suffice to make the patient fairly comfortable. During the first few days we may administer small doses of calomel and bismuth, and later dilute hydrochloric acid, pineapple juice and some good wine or cognac. Hexamethylenamine (2 to 5 grains) is useful during the entire course of the disease. In intestinal hemorrhage, an ice coil to the abdomen and opium suppository ($\frac{1}{10}$ grain for every year of the child's age) will be found very efficient. When the hemorrhage is excessive, transfusion and surgical treatment should be instituted without delay. Rest in bed should be enjoined for at least two weeks after defervescence. The diet should be fluid (milk with tea, amply sweetened with milk sugar, or malt sugar, soups, light gruels, chicken broth, zoölak, egg with sherry wine, ice cream) during the acute course of the disease, and semisolid thereafter, care being taken not to overfeed. Transition to a more solid diet should be very gradual. Relapses call for the same mode of treatment as the original attack. During convalescence the different bitter tonics and iron are very desirable, and a sojourn at the seashore often works wonders.

Complications should be carefully guarded against and immediately treated according to indications. Frequent change of position of the patient is usually effective to prevent serious pulmonary complications as well as decubitus. The skin should be hardened by alcohol, alum water, etc., and as much as possible protected by air cushions. The slightest abrasion of the skin should at once be treated by antiseptic

dressings (2 per cent solution of aluminum aceticotartrate). It is claimed that the external application of castor oil prevents and cures decubitus. Insomnia and excessive restlessness sometimes require hypnotics.

Typhus Exanthematicus

TYPHUS FEVER, SPOTTED FEVER, SHIP FEVER, JAIL FEVER, CAMP FEVER, TABARDILLO (MEXICO)

Typhus fever is an acute infectious, endemic, epidemic and sporadic disease of doubtful origin (the B. typhi exanthematici, Plotz, is as yet not generally accepted as the true cause), transmitted through the body louse and characterized by a discrete, maculated, petechial rash, and moderate fever, terminating by crisis in from ten to fourteen days. The prodromic stage lasts from a few hours to several days and is followed by severe headache, usually frontal, and pain in the back and extremities. The eruption generally appears on the fourth or fifth day, is rose colored or hemorrhagic, and scattered all over the body and more especially over the trunk and limbs. The spots do not disappear on pressure. The patients usually manifest a tendency to very rapid breathing, in the absence of other lung symptoms. In young children bronchopneumonia is not uncommon. The blood shows a marked leucocytosis. During the absence of an epidemic the diagnosis is often difficult until the termination of the disease (the sudden drop of temperature!) and may readily be mistaken for typhoid fever (Widal positive, see p. 86) and relapsing fever (recurrence of fever, spirillum in the blood). Positive Weil-Felix reaction (*q. v.*) is decisive of the diagnosis of typhus exanthematicus.

The treatment is chiefly prophylactic (destruction of lice, fleas, etc.) and hygienic. Individual symptoms are treated according to indications.

Typhus Recurrens

(FEBRIS RECURRENS, RELAPSING FEVER, SPIROCHETOSIS)

This affection is quite common in Europe, and in some African states, but is very rarely observed in the United States. It is due to a spirochete, varying in type in different countries, which was first described by Obermeier in 1873. Other types of the spirochete have since been demonstrated by Dutton, Carter and Novy. The disease is conveyed to men by ticks, bedbugs, fleas, lice and flies. It is characterized by two or more febrile paroxysms of six days' duration succeeded by afebrile intervals of equal length. The temperature ranges between 104° and

106° F. and comes down by lysis with profuse sweating and sound sleep. During the afebrile stages the patient seems in fairly good health. There is usually an enlargement of the liver and spleen and in severe cases profuse diarrhea, dysentery and hematemesis are observed. The spirochetes are circulating in the blood during the height of the fever.

Treatment.—Prophylaxis is readily accomplished by extermination of the purveyors of the disease. The active treatment consists of intravenous administration of neosalvarsan in doses from 0.1 to 0.4, to be repeated every three or four days until the spirochete has been eliminated from the blood. Other symptoms are treated according to indications.

Glandular Fever

(Pfeiffer)

Glandular fever is an infectious disease which sometimes occurs in epidemics, most frequently among children from two to eight years of age. The portal of entry of the infection is the rhinopharynx. Simultaneously with a rapid rise in temperature (102° to 104° F.) there appear more or less painful swellings of the submaxillary and cervical glands—which usually interfere with the movements of the head—redness of the throat, headache, sometimes vomiting and diarrhea, and occasionally enlargement of the spleen and liver.

The fever usually disappears soon, sometimes within twenty-four hours ("one day fever"), but the glandular swelling persists for several weeks and exceptionally spreads to other lymph nodes of the body, e.g., bronchial (cough), esophageal (dysphagia) and retroperitoneal (pain in the abdomen, especially on pressure). Occasionally this disease is complicated by nephritis, but the prognosis as a whole is good.

In the early stages glandular fever may be mistaken for tonsillitis or parotitis.

The treatment is symptomatic, calomel and the salicylates internally and a mild iodine ointment externally, ordinarily serving the purpose of relieving the pain, fever and swelling. Tonics and change of air in protracted cases.

Malaria

(Febris Intermittens, Febris Remittens, Estivo-Autumnal)

Malaria is endemic in the greater portion of the inhabited world, and is most prevalent in swampy tropical regions. No age is exempt from this disease. The exciting cause of malaria is the hematozoön

of Laveran conveyed to the human body principally by the bite of the Anopheles mosquito which has previously sucked the blood of a malarial patient and has acted as an intermediate host for the malarial parasite. The hematozoön enters the blood corpuscles and, after undergoing the different stages of development, the blood current—at this time giving rise to the characteristic chill or paroxysm. Varying with the period of maturity and the species of the plasmodium, the febrile attack may occur every day (quotidian); every two days, going on the third (tertian); every three days, going on the fourth (quartan) day; or may be more or less continuous with daily remissions (remittent or estivo-autumnal fever). Furthermore, several types of plasmodia or several generations of the same parasite may circulate in the blood, and, by varying in the period of their maturity, may give rise to double tertian or quartan paroxysms daily or every other day at different hours.

Intermittent Fever

This form of malaria is characterized by the occurrence, at regular intervals, of paroxysms divided into four stages—premonitory, chill, fever, and the sweat. During the premonitory stage the patient complains of headache, lassitude, and nausea; he vomits, yawns, is irritable and drowsy. Suddenly he is seized with a feeling of cold—the chill. The features become pinched, the lips blue, the skin cool and rough (*cutis anserina*); he shivers and shakes, and his teeth chatter while the thermometer in the axilla or rectum shows a decided rise of temperature. These phenomena may continue for from a few minutes to an hour or longer and are then gradually replaced by those of the hot stage, *i. e.*, hyperpyrexia, flushed face, headache, full pulse, intense thirst, scanty urine, sometimes nausea, vomiting and severe nervous manifestations. The hot stage lasts from three to six hours or longer, and subsides gradually, being succeeded by more or less marked sweating, defervescence and rapid abatement of the other symptoms. The duration of the entire paroxysm is from six to twelve hours, after which time the patient is apparently well—until the return of a new attack which as already mentioned may occur every day, every two days or three days.

This description corresponds with the symptomatology of typical intermittent fever, uninfluenced by medication, as it occurs in children over ten years of age. It is thus identical with that in adults. In younger children the course of the paroxysms presents numerous deviations. The prodromic and cold stages may be absent or of very
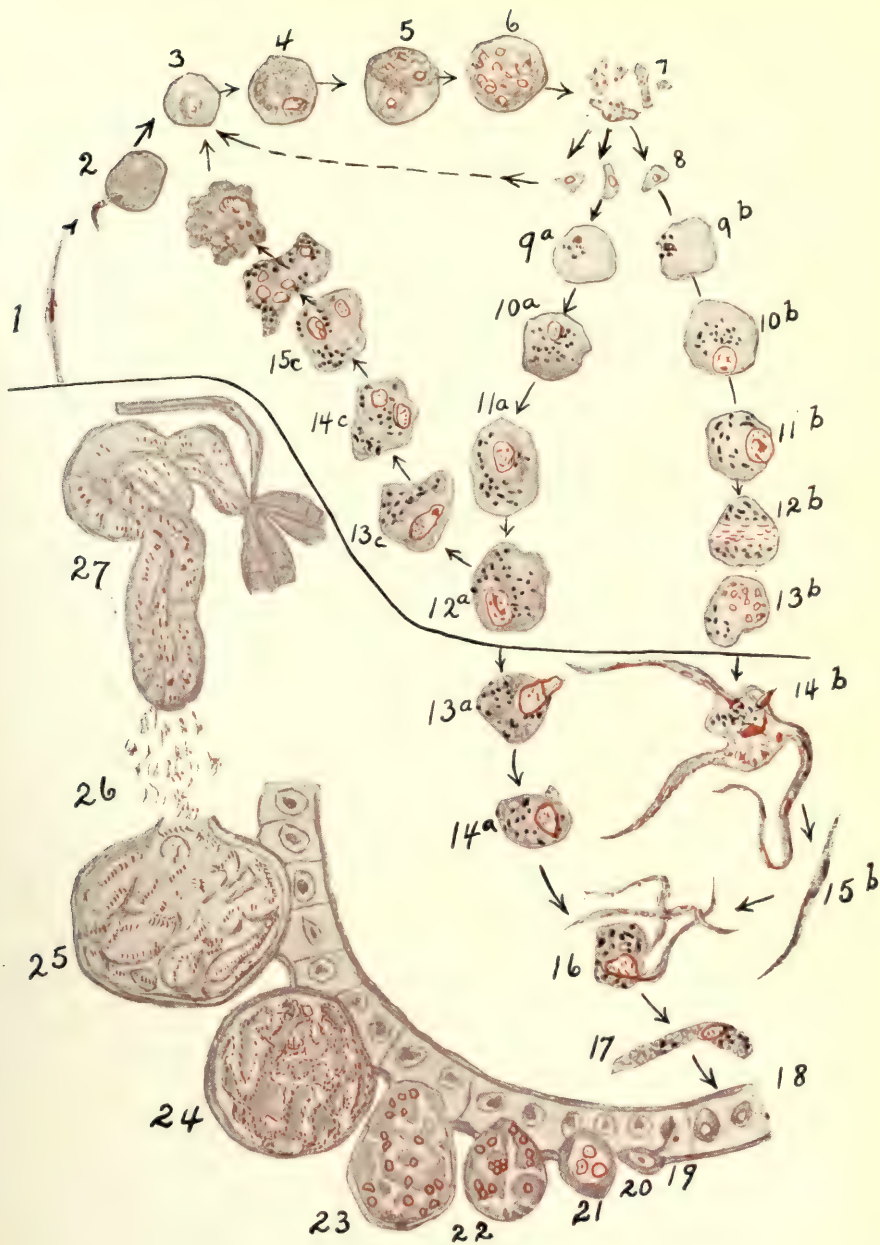
PLATE IX

LIFE-CYCLE OF PLASMODIUM VIVAX

(After Grassi and Schaudinn)

The human cycle is above the transverse line, some rearranged by Kissalt and Hartmann. The cycle in the mosquito is beneath. 1 to 7, Schizogony; 1, sporozoite; 2, entrance of sporozoite; 3 and 4, growth of the schizont; 5 and 6, nuclear division of the schizont; 7, formation of the merozoites; 8, merozoites; 9a to 12a, growth of the macrogametocyte; 9b to 12b, growth of microgametocyte; 13c to 17c, parthenogenesis of the macrogametocyte; 13a and 14a, maturation of macrogamete; 13b and 14b, growth of the microgamete; 15b, microgamete; 16, fructification; 17, Ookinete; 18 to 20, entrance of the Ookinete into the stomach wall of the mosquito; 20 to 25, sporogony; 22 and 23, nuclear multiplication in the sporont; 24 and 25, formation of the sporozoites; 26, passage of the sporozoites to the salivary gland; 27, salivary gland of the mosquito with sporozoites (Magn. 1 to 17c, 1200 to 1; 18 to 27c, 600 to 1.) Park: *Pathogenic Bacteria and Protozoa.*

brief duration. The chill may be replaced by grave nervous manifestations, such as convulsions, or be indicated only by cyanosis of the lips and the tips of the fingers and toes. Sweating is slight or absent, or may be well marked and continue until the subsequent paroxysm of fever. Young children are rarely entirely free from discomfort during the intermittent stage. As a rule, they are exhausted, restless, have no appetite, etc. With repeated attacks of the fever there is marked swelling of the spleen and great diminution in the number of red blood cells.

In view of the aforementioned deviations from the typical course of the paroxysms, the diagnosis of intermittent fever in young children often presents great difficulties. It is apt to be mistaken for tuberculous (meningitis, lymphangitis, peritonitis, etc.) and pyemic (empyema, pyelitis, ulcerative endocarditis, otitis, etc.) processes, typhoid and influenza. A correct diagnosis, however, can usually be arrived at by exclusion, always bearing in mind the facts that in malaria the plasmodium malariæ or secondary pigmentation of the blood cells is invariably present in the blood and that the course of the disease is greatly modified by full doses of quinine.

Remittent (Estivo-autumnal) Fever

This type of malarial fever is usually observed in the temperate zones, principally in the autumn. In institutions where large numbers of children are congregated, it may occur in epidemic form and lead to grave diagnostic errors. It usually sets in suddenly with malaise and chilliness, followed by fever with exacerbations and remissions, the temperature during the latter, however, remaining constantly above normal. The other symptoms are very indefinite. As in all febrile diseases, anorexia, nausea, sometimes vomiting, headache, drowsiness and lassitude predominate. In some cases gastrointestinal symptoms prevail, in others respiratory. But the cardinal manifestations of the affection are the continued fever of from one to three weeks' duration, with irregular remissions, palpable spleen, and the plasmodium malariæ in the blood. Bearing these clinical symptoms in mind and those of the diseases suspected, there ought to be no difficulty in differentiating remittent fever from typhoid fever or protracted influenza—with both of which diseases it is most apt to be confounded. The quinine test is not reliable in the remittent form of malaria as the fever often resists medication.

The prognosis of remittent fever is favorable, except for the tendency to recurrences at shorter or longer intervals and of ultimately becoming chronic.

Chronic Malarial Cachexia

The diagnosis of this condition is often very difficult, since its principal symptoms—anemia, debility, enlarged spleen and liver—are also pathognomonic of severe rachitis, pseudoleukemia, and similar wasting diseases. Corroborative data may be obtained from a history of previous attacks of either intermittent or remittent fever or the occurrence of periodical headache, neuralgia, dysentery or hematuria. One should be very cautious, however, in making a hasty diagnosis of "malaria" unless there be ample reason for exclusion of the other affections and the therapeutic quinine test prove positive.

Chronic malarial cachexia *per se* is not dangerous to life, but is apt to prove so from its concomitant symptoms, such as profound anemia and amyloid degeneration of the viscera.

Treatment.—As malarial fever is ordinarily contracted through the bites of mosquitoes, to prevent malarial disease, we must either destroy the mosquitoes or avoid their bites. An effort should be made also to isolate, by mosquito netting, all cases of acute malarial disease, in order to deprive the mosquitoes of the infective material. Another very important measure is to prevent the breeding of mosquitoes. Mosquitoes lay their eggs in water barrels, pans, tin cans, pots, kettles, wells, springs, rain pools, cess pools, drainage taps, ponds—in short, wherever stagnant water is found. We have to see to it that all water receptacles are closely covered with thin wire gauze, and that where drainage cannot be carried out, the surface of ponds, etc., are covered with a film of kerosene oil. One ounce of oil to 15 square feet of water will usually suffice. The oil must be renewed about once a week during the mosquito season. A solution containing 1 pound of sulphate of copper and 1 pound of unslaked lime in 10 gallons of water will kill the mosquito larvæ when added in proportion of 1 of the solution to 50 of the infected water.

White people settling in malarial tropical regions should not plant their houses near native settlements.

Where the aforementioned prophylactic measures cannot be properly enforced, resort should be had to the routine administration of quinine during the mosquito season. Whether as a prophylactic or curative measure, quinine is the specific destructive agent of the malarial parasites. To obtain prompt results it should be given in full doses. Children tolerate relatively much larger quantities of quinine than adults. An infant of two years requires about 15 or 20 grains a day until the attack is controlled, and smaller doses after. For children unable to take quinine in capsules, I prefer the newer "tasteless"

quinine preparations, such as quinine ethyl carbonate, diquinine carbonic ester, etc., or quinine tannate, or I administer the ordinary bitter quinine per rectum (10 grains of quinine subsulphate in 4 drams of water by means of colon tube). In cases of marked gastric irritability or in those very grave in nature or protracted in course, quinine may be employed in 5 grain doses hypodermically. For this purpose, bimuriate of quinine and urea, the hydrochlorosulphate, the hydrobromate, or the bisulphate may be used. Ugly sloughing which is apt to follow at the site of the injection may be prevented by cleanliness of the needle and skin, and by throwing the solution deeply into the subcutaneous tissues and sealing the point of puncture with adhesive plaster.

In protracted cases iron and arsenic (Fowler's solution) will be found useful additions to the quinine. When there is a great tendency to recurrences of the malaria, permanent residence in dry mountainous regions will sometimes remain the only curative measure at our command.

℞ Quinine Ethyl Carbonate, or
        Diquinine Carbonic Ester   ℨ ss | 2.00
     Syr. simplicis   ℥ ii | 60.00
     M.
     S.—One teaspoonful every two to four hours, for
a child three years old.

℞ Quininæ Mur.   gr. xv | 1.00
     Acetanilidi   gr. vi | 0.40
     Podophyllini   gr. ⅛ | 0.008
     Ext. Nucis Vomicæ   gr. ¼ | 0.016
     M. ft. caps. no. xii.
     S.—Two capsules every three hours, for a child six
years old.

℞ Acidi Arsenosi   gr. ¹⁄₁₀ | 0.006
     Quininæ Mur.   ℨ ss | 2.00
     Ferri Sulph. Exs.   gr. x | 0.66
     Pulv. Rhei   gr. v | 0 33
     M. ft. caps. no. xx.
     S.—Two capsules every six hours, for a child ten
years old (in chronic malaria).

℞ Elixir Ferri Pyrophosphatis, Quininæ
        et Strychninæ (N.F.)   ℥ i ss | 45.0
     Syr. Aurantii   q. s. ℥ iii | 90.0
     M.
     S.—One teaspoonful three times a day, for a child
four years old (in convalescence).

Treatment should not be discontinued until the blood has become free from plasmodia or pigment and the spleen has assumed its normal size.

Dengue

(BREAKBONE FEVER. SEVEN-DAYS-FEVER)

This specific affection is transmitted by bites of mosquitoes, the *Culex fatigans* and *Stegomyia fasciata*. While most common in the tropics, it is not rarely observed in Texas. It is characterized by two febrile paroxysms of about three days' duration with an intermission of a day or two. The drop of temperature is accompanied by profuse sweating. With the second rise of temperature a roseolar or scarlet-like eruption makes its appearance. The disease is associated with a marked leucopenia, severe pain in the head (eyeballs), back and joints, and in young children, delirium and convulsions—the latter probably the result of hyperpyrexia (104° to 106° F.). The second or third attack is milder than the first one. The disease is benign in character and usually responds promptly to the administration of salicylates and quinine.

In a recent paper on dengue Ch. F. Craig* reaffirms his views on the similarity of dengue and yellow fever, both clinically and etiologically and speaks strongly in favor of the spirochetal nature of dengue. Clinically, both diseases have a sudden onset, run a comparatively rapid course, and terminate by crisis rather than by lysis.

In both diseases the cause is present in the blood, but only during certain periods; in both, the injection of unfiltered blood from patients suffering from the disease results positively, the incubation period in yellow fever being usually three and a half days, while in his experiments, the incubation period in dengue averaged three days, fourteen hours; in both, the injection of filtered blood produces the disease, thus proving that both are due to a filtrable virus; in both, the transmitting agent is a mosquito; and both, finally, have proved to be non-contagious.

Rocky Mountain Fever

(TICK FEVER, SPOTTED FEVER)

This disease is endemic in the valleys of the Rocky Mountains in Idaho and Montana. It has also been found in the valleys of Nevada and Wyoming. It occurs in the spring months and while the cause of the affection is still unknown, it has been definitely established, especially by Ricket and King that it is transmitted by infected ticks, the Dermacentor occidentalis. The disease is characterized by a continu-

*Jour. Am. Med. Assn., Oct. 30, 1920.

ous moderate fever, which falls by lysis, severe chills, arthritic and muscular pain, and a profuse macular, or petechial rash, which appears (from the second to the fifth day) first upon the ankles, wrists and forehead but soon spreads over the entire body. In severe cases there may be delirium, tachycardia, out of proportion to the temperature, albuminuria and casts and jaundice.

Treatment.—Protection against the bites of the ticks, particularly by protecting the hands and feet. The active treatment is symptomatic.

Pestis Americana
(Yellow Fever. The Yellow Jack)

While the specific microbe of this acute infectious fever is still un-discovered,[*] it is definitely settled—thanks principally to the investigations of Ch. Finley, Reed, Carroll, Agramonte and Lazear—that yellow fever is spread by the bite of the *Stegomyia calopus* mosquito.

Pathology.—The liver is the chief seat of the pathologic alterations. The liver cells swell and degenerate and by pressure upon the capillaries obstruct the flow of bile and thus give rise to the hepatogenous jaundice. The degenerative process proceeds also in the interlobular capillaries, interfering with the portal circulation and causing congestion of the gastrointestinal tract. On postmortem examination the stomach and intestines are often found to contain large quantities of blood. Puncti-form hemorrhages are observed also in the other organs of the body.

Symptomatology.—After an incubation period of from 2 to 6 days, the attack usually sets in with vomiting, severe abdominal and spinal pain, high fever (about 104° F.) not rarely convulsions, and albuminuria (usually the second day). This attack may last from two to four days and is followed by a 24 hours' remission accompanied by sweating, when the second paroxysm of fever develops with marked jaundice, hemor-rhage from the stomach (black vomit), slow pulse and general prostra-tion. This paroxysm in favorable cases usually lasts from two to three days; the patient passes into a sound sleep and is then well on the road to convalescence. In unfavorable cases the temperature continues to rise, the hemorrhagic vomiting persists, and there develop in addition, clammy sweats, complete anuria, delirium, convulsions and coma. In the United States the mortality ranges between 20 and 25 per cent.

Mild cases may be mistaken for dengue and malarial fever—in neither of these affections, however, do we find albuminuria and marked jaundice. Furthermore, malaria presents the plasmodium in the blood. (See ''Dengue.'')

*Noguchi claims that it is a spirochete, the Leptospira icteroides. (Jour. Am. Med. Assn., Jan. 8, 1921.)

Treatment.—Destruction of mosquitoes at their source; screening of the patient, and fumigation of the sick room with sulphur or formaldehyde. Prophylactic inoculation is recommended by Noguchi and Pazeja.

The active treatment is symptomatic. With the claim of a spirochete being the cause of yellow fever, neosalvarsan would seem to me to be the remedy worth trying. Plenty of alkaline waters, warm baths, liquid diet, cardiac stimulants, and sedatives, if the pain is very severe.

Ileocolitis Epidemica

(DYSENTERY)

This form of dysentery is entirely distinct from hemorrhagic enteritis or proctitis spoken of in connection with gastroenteritis on page 257. It is an infectious epidemic, and sometimes sporadic disease, caused by the dysentery bacilli described by Shiga, Kruse and Flexner. Amebic dysentery which is seen here sporadically is endemic in the tropics. The lesion is localized principally in the sigmoid flexure and rectum, or also in the entire colon up to the ileocecal valve or even the lower portion of the ileum, and varies from a simple inflammation of the mucosa to a croupous, diphtheritic inflammation, with a fibrinous exudate or a membranous deposit, ulcer formation, and necrosis (gangrene). Dysentery is most common during August and September and late in autumn. It most frequently affects young children who are on a mixed diet.

In the majority of instances dysentery begins with simple diarrhea, without constitutional symptoms, and after from twenty-four to forty-eight hours is followed by the characteristic symptoms later to be spoken of. In some cases the onset is sudden with high fever and, in small children, with convulsions. Once the affection is established the symptomatology is quite pathognomonic: colic, tenesmus, and bloody stools. The colic precedes and accompanies defecation and is followed by severe and prolonged tenesmus. The bowel movements vary between ten and thirty or more in twenty-four hours, and the dejecta consist either of pure blood or of blood and dirty ragged shreds of tissue and fecal masses. The abdomen is most frequently sunken, permitting palpation of the contracted colon. The tongue is dry and heavily coated, the lips are cracked and covered with sordes, the appetite is lost, and the child suffers from intense thirst, and occasionally from nausea and vomiting. As a rule, the temperature is raised (intermittent), but it may be normal or subnormal. After a few days the patient becomes greatly emaciated and prostrated, very anemic, and the expression of the face denotes great suffering. Quite a number of children succumb during this stage of the disease (fulminating type); others again continue to battle for life and after a course of from seven to ten days begin to improve, the stools

becoming less bloody and more feculent in character, the anorexia less marked, and the general condition much better. Relapses are not rare, and when they occur, there is a great tendency toward the transition of the acute into a chronic process, with a very tedious convalescence, or death from exhaustion.

An attack of dysentery may be complicated by perforation peritonitis, abscess of the liver, fissura or prolapsus ani, pulmonary affections, noma, parotitis suppurativa, etc., and may be followed by intestinal cicatrices and stenosis, paralysis of the sphincters, paresis of the extremities, and marasmus.

The very protracted cases of dysentery are usually found to be due to the ameba coli (entameba histolytica dysenteriæ). The differentiation between this form of dysentery, that due to Shiga's bacillus, and catarrhal enteritis is important from the therapeutic point of view and can readily be made by a bacteriologic and microscopic examination of the dejecta. Furthermore, it is well to remember that foreign bodies in the lower bowel may give rise to a group of symptoms similar to those of dysentery; and that an inflamed prolapsed rectum, intussusception, an ulcerated rectal growth or hemorrhoids with coincident enteritis are very apt to mislead in the diagnosis. Careful examination (inspection and palpation) of the rectum disposes of these difficulties.

Treatment.—Similar to a patient with typhoid, patients suffering from dysentery need not be strictly isolated. The dejecta and everything coming in contact with them, however, should be thoroughly disinfected. During an epidemic the drinking water, fruit and vegetables should be boiled, and all modes of exposure to infection (mosquitoes, flies!) avoided.

Acute dysentery calls for perfect rest in bed, an opiate (preferably hypodermically or per rectum) for the relief of pain, and light astringent diet (tea and toast without sugar, rice and barley gruel without milk, and later albumin milk with equal parts of barley or rice water). In the beginning the bowels should be cleansed with a moderate dose of castor oil or syrupus rhei by mouth and one sterile cool water irrigation. The patient is then put on pulveris Doveri, $\frac{1}{8}$ grain, for every year of the child's age every three hours, and if there is no vomiting also on the following mixture:

℞	Bismuthi Subnitratis		℥ iv	15.00
	Vini Ipecacuanhæ		℥ i	4.00
	Mist. Creatæ Comp.		℥ iv	15.00
	Aq. Anisi	q.s.ad f ℥ iii		90.00
	M.			

S.—One teaspoonful every two to four hours, for a child three years old.

In severe cases the intestines should be irrigated once a day with 1:1000 of nitrate of silver, and once a day with 1:1000 quinine sulphate solution, the latter especially in amebic dysentery. The irrigation should be executed *very gently* by means of a soft rubber catheter attached to an ordinary irrigator. Sometimes starch water (1 ounce to 1 pint) with a few drops of tincture of opium will relieve the tenesmus. Hydropathic applications to the abdomen (plain Priessnitz compress, or warm turpentine stupes) are useful.

Flexner* recommends polyvalent antidysenteric serum (10 to 20 c.c.) subcutaneously or intravenously.

Collapse should be combated by local heat, cognac, red wine with a hot infusion of cinnamon, camphor, strychnine, etc. During convalescence care in dieting is still demanded (recurrences are common), and the persistent anemia calls for iron, analeptics in the form of strengthening food (fresh eggs, milk with cereals, broths, etc.) and plenty of fresh air, and, whenever possible, a sojourn in the country, preferably at the seashore.

In amebic dysentery quinine (2 to 5 grains t. i. d.) by mouth, and emetine hydrochlorate ($\frac{1}{8}$ grain) hypodermically once a day are of great service.

In chronic dysentery the tannates in conjunction with the quinine and silver irrigations do better than the bismuth preparations. Otherwise the management is the same as in acute dysentery. The more protracted the course, the greater the exhaustion and loss of blood; and the younger the child, the worse the prognosis. The mortality in different epidemics varied between 5 per cent and 30 per cent. Early attention is a very great factor in reducing the mortality and the tendency toward chronicity.

Rheumatismus Acutus

(Rheumatic Fever, Polyarthritis Acuta)

Acute inflammatory rheumatism is an infectious disease with a specific predilection for the fibrous tissues and serous membranes. The muscular and neural structures, however, are not exempt from it. The discovery of the rheumatism-producing microorganism is a matter probably of the very near future. In fact it is quite probable that the so-called streptococcus or diplococcus rheumaticus which is frequently found in the exudate of the joints and in the blood plays a very important rôle in the causation of rheumatic fever.

Rheumatism is most common in children over five years of age, but no age (even infants under one year) is exempt. A hereditary disposition can usually be traced in the majority ot cases.

*Jour. Am. Med. Assn., lxxvi, No. 2, 1921.

Similar to other infectious diseases, rheumatic fever is most prevalent in certain climates and seasons of the year. It presents a prodromic stage of variable duration, which is characterized by chilliness, languor, etc. Like the eruptive fevers it is manifested by general febrile disturbance with local lesions. To a certain extent it is self-limited, since with exhaustion of the fertile soil in one place, the inflammation "jumps" to another place. It ordinarily yields promptly to specific medication; in this respect also resembling infectious fevers, e. g., malarial fever.

After a brief prodromic stage, the symptoms of acute rheumatism usually set in suddenly, with chills, rise of temperature, vomiting, and vague pain in several parts of the body. In very young children the onset is not rarely associated with cerebral symptoms, especially convulsions. Older children often complain of sore throat (lacunar tonsillitis), and in some cases articular swelling forms the first principal manifestation of the affection. The disease, once established, differs in its symptomatology and course but little from that observed in rheumatism in adults, except, as will be seen later, that in children there is a greater tendency toward cardiac complications, while the articular involvement is usually less pronounced.

The joints of the knee, ankle, elbow and wrist are most commonly affected, occasionally also those of the phalanges and hip. In one case under observation the lower dorsal vertebræ were so severely affected as to greatly resemble acute spondylitis. The articular involvement is accompanied by stiffness, slight redness, swelling and excruciating pain, the latter especially on attempting to walk, or moving or handling the parts affected. The inflammation may abruptly cease at one or more joints and, as suddenly, attack others. During the acute stage the temperature varies between 102° and 104° F., and as the inflammation "jumps" from joint to joint there is usually a sharp rise of temperature. Correspondingly, the temperature falls with abatement of the local manifestations. The urine is usually scanty and high-colored, filled with urates, and occasionally contains traces of albumin. The characteristic sour (lactic acid) sweats observed in adults are much less pronounced in children.

There is no definite limitation to the duration and course of the affection. Mild cases, after pursuing a mild febrile course for a few days, may either recover entirely or enter into a subacute, afebrile stage, which for weeks and months may be manifested by vague articular and muscular pain, and ultimately end either in complete recovery, or leave behind some form of subacute or chronic heart dis-

ease. Indeed, it is usually in such cases that the heart affection is overlooked, and unexpectedly discovered some time (years!) later, without being able to disclose a rheumatic history. Severe cases may run a febrile course of from three to five weeks and sometimes as many months, if left untreated. It is well to remember that the gravity of an attack is not always commensurate with the severity of the articular involvement. In quite a number of cases, endocarditis or pericarditis, or both, may predominate while the other symptoms are barely noticeable. Hence the importance of a routine and careful examination of the heart of children suffering from rheumatic and "growing" pain, or chorea. The latter disease, by the way, is closely allied to, and may precede, accompany or follow rheumatism in its various forms. (See "Chorea".)

The earliest symptoms of rheumatic endocarditis are increase of frequency and intensity of the heart beat and precordial pain. This is soon followed by the usual physical signs of endocarditis—those of mitral regurgitation predominating. Endocarditis forms the most frequent (in about 60 per cent) complication of inflammatory rheumatism and usually sets in within the first ten days from the onset.

Pericarditis is observed only in about 10 per cent of the cases, and somewhat later than endocarditis. It is manifested by a dry friction sound, heard at the apex or base of the heart, or by a serous exudation which may rapidly, and unnoticeably, disappear, or persist and lead to pericardial adhesions and their accompanying more or less grave sequelæ.

Less frequent complications are pleuritis and pneumonitis. Both these affections are ordinarily limited to the left side. The pleuritic effusion may be serous or serofibrinous and is most frequently associated with pericarditis. Of still less frequent occurrence are peritonitis and nephritis. The abdominal pain, however, not infrequently complained of by children during an attack of rheumatism, is usually due to muscular hyperesthesia and not to peritoneal involvement.

As in adults, rheumatism of children may also affect the muscles. Rheumatic torticollis is especially common, and in severe cases is apt to be mistaken for cervical spondylitis. Muscular rheumatism affecting the muscles of the lumbar region may resemble lumbar spondylitis; and that of the leg may give rise to symptoms (pain on motion, lameness, stiffness, etc.) simulating coxitis, or poliomyelitis. As previously mentioned, rheumatism of the abdominal muscles may simulate peritonitis, while rheumatism of the intercostal muscles may be mistaken for dry pleurisy. In all these cases a diagnosis can usually be

arrived at by bearing in mind the pathognomonic symptoms of the affections the muscular rheumatism resembles, and the fact that the latter promptly yields to the salicylates, and that as a rule, there is a history of involvement of other groups of muscles.

Rheumatism may also affect the periosteum and give rise to thickening of the underlying bone which condition with the accompanying pain and fever, may simulate incipient osteomyelitis. From what has

Fig. 93.—Rheumatic torticollis of several weeks' duration in a child six years old which greatly resembled cervical spondylitis.

been said, it can readily be seen that the diagnosis of rheumatism in its various phases is far from being easy.

Moreover, articular rheumatism may also be mistaken for syphilitic, gonorrheal, tuberculous, and the so-called septic arthritides, scurvy and its allied affections.

In our endeavor to differentiate rheumatism from the divers forms of articular and periarticular inflammations, we must bear in mind

that rheumatism is a primary febrile affection, as a rule, sudden in development; that its inflammatory process is transient, and its localization multifarious and rapidly shifting, and, finally, that its course is promptly and often permanently influenced by the salicylates.

Differential Diagnosis

Epiphysitis Syphilitica.—Syphilitic epiphysitis develops slowly, in the first few months of life—rather exceptional for rheumatism—in association with other symptoms of congenital syphilis. It runs an afebrile course and yields promptly to antisyphilitic medication.

Arthritis Heredosyphilitica (Tarda).—Hereditary syphilitic arthritis develops gradually, and affects principally one or both knees. It is usually associated with other syphilitic symptoms, especially interstitial keratitis. As a rule, the subjective disturbances are incongruous with the severity and extent of the local signs, and the arthritis is but rarely accompanied by inflammatory symptoms. It yields promptly to antisyphilitic medication. Puncture of the swelling reveals serofibrinous fluid and not rarely the spirochete.

Arthritis Gonorrheica.—Gonorrheal arthritis occurs as a complication of gonorrheal ophthalmia, urethritis, or vulvovaginitis. It is most frequently limited to one knee, more rarely to both knees, or to the maxillary or sternal articulations, and is accompanied by pronounced inflammatory local and general symptoms. The articular involvement is more lasting than that of acute rheumatism, and resists antirheumatic measures.

Arthritis Tuberculosa.—Tuberculous arthritis develops gradually, usually remains limited to one joint, and resists antirheumatic treatment. Atrophy of the affected limb sets in early, and an x-ray examination often shows involvement of the bone. The tuberculin reaction is often positive.

Arthritis Septica.—Septic or infective arthritis is usually monoarticular and arises secondarily to sepsis (*e. g.*, purulent arthritis in sepsis neonatorum) or to acute infectious diseases, such as typhoid fever, influenza, pneumonia, diphtheria, scarlatina, etc. In two cases under our observation purulent arthritis of the knee followed tonsillectomy. The history is the most reliable clue in the diagnosis, and the finding of the streptococcus, pneumococcus, etc., in the seropurulent fluid obtained by exploratory puncture of the swelling is decisive.

Scorbutus (Barlow's disease) purpura hemorrhagica and hemophilia (with sanguineous effusion into the joints) also may be mistaken for acute articular rheumatism. In the hemorrhagic diseases, however, there are hemorrhages from and into other parts of the body. The

articular swelling is not as evanescent. Fever is usually absent or slight. Furthermore, Barlow's disease is observed in very young infants, who are rarely attacked by rheumatism, and yields promptly to antiscorbutic diet. Antirheumatic treatment is futile.

Osteomyelitis.—The swelling does not appear until a few days after the onset of the disease, and has its center, not opposite the joint, as in articular rheumatism, but above or below, opposite one or other of the epiphyses of the bones entering into the formation of the joint. In advanced cases the swelling extends along the shaft to a variable distance. In contrast to osteomyelitis rheumatism is rarely limited to a single joint, and its swelling never suppurates. Leucocytosis is absent in rheumatism, and, as a rule, marked in osteomyelitis. A skiagraph is helpful in the differential diagnosis.

Prognosis.—Rheumatic fever *per se* is very rarely fatal, but only very few patients emerge uninjured from a severe attack of rheumatism. In probably two-thirds of the cases some form of heart disease is acquired, which sooner or later manifests evidence of its destructive character. This obtains particularly in recurrent rheumatism, as well as in cases improperly cared for, as regards rest and specific medication.

Treatment.—Rest in bed is the most important therapeutic measure in the prevention of grave complications and sequelæ, and should be enjoined at least during the febrile course of the disease. Medicinally, the salicylates act specifically in all acute rheumatic conditions, and their administrations should be continued until every vestige of the disease has disappeared. In the beginning, the salicylates should be pushed to their full tolerance—let us say 1 grain of the sodium salicylate for every year of the child's age, every two hours, until the acute symptoms have been arrested, then every three or six hours according to indications. The salicylates may be alternated with sodium bicarbonate until the urine becomes alkaline, or with atophan. With the appearance of cardiac complications, the iodides, in small doses, should be added, and if necessary, also digitalis. For the relief of articular pain and swelling, the joint should be enveloped in absorbent cotton wrung out of a saturated solution of bicarbonate of soda. The compress should be covered with oiled silk and a flannel bandage and changed every two to four hours. When the pain is very acute I have found the following very serviceable:

℞ Olei Gaultheriæ
   Guaiacolis
   Ichthyolis   āā ℥ ss | 2.00
   Adipis Lanæ   ℥ i | 30.00
      S.—Apply gently twice a day, and
      cover with a flannel bandage.

Acute rheumatism being an infectious disease, I have no faith in "mathematical dietetics" as a cure of the disease; hence, do not employ any specific dietary, but limit the diet to a so-called "fever diet" during the febrile stage of the disease and to easily digestible food of all sorts later. This has the advantage of maintaining the nutrition of the patient who at best is weak and anemic. On the other hand, in a number of refractory cases the diet has to be limited, and I have found that a purely vegetable and cereal diet (without milk or sugar, but made palatable by the addition of sweet butter) will often be very beneficial. The prolonged use of the iodides and cod liver oil is always in order in the convalescent stage, and a sojourn in a dry and high inland resort will prevent recurrence and chronicity.

℞	Natrii Salicyl	℥ ii	8.00
	Mist. Rhei et Sodæ	℥ iii	12.00
	Aq. Destil.	q. s. ad f ℥ iii	90.00
	M.		

S.—One teaspoonful every two to four hours, for a child four years old.

℞	Antipyrinæ	℥ ss	2.00
	Natrii Salicyl.	℥ iss	6.00
	Caffeinæ Natrii Benzoatis	gr. xvi	1.00
	Syr. Simplicis	℥ iv	15.00
	Aq. Destil.	q. s. ad f ℥ ii	60.00
	M.		

S.—One teaspoonful every six to twelve hours, for a child four years old (for quick relief of pain.)

| ℞ | Olei gaultheriæ | ℥ i | 4.00 |
| | Ft. caps. no. xii. | | |

S.—One capsule every four to six hours, for a child six years old (for subacute rheumatism).

The throat should be kept disinfected by Dobell's or similar antiseptics. Constipation should be remedied by cascara sagrada.

Recurrent rheumatism often calls for complete enucleation of the tonsils, and careful attention to the teeth.

Rheumatoid Arthritis

(RHEUMATISMUS CHRONICUS, ARTHRITIS DEFORMANS)

Chronic rheumatism in children is very rare. Similar to what occurs in adults, it may supervene after recurrent attacks of acute or subacute rheumatism, or, very exceptionally, it may develop primarily. In either case the local manifestations are clinically alike, and consist

of gradual enlargement of the affected joints, with atrophy of the muscles around the joints, painful and hindered motility, ankylosis, and deformity of the bones at the articulations. It is usually bilateral. The course of this form of rheumatism though very protracted, and extending over a period of years, is usually not as slow as in adults. It eventually leads to crippling of the patients, and fatal termination either from exhaustion or complicating tuberculosis.

Chronic articular rheumatism may be confounded principally with syphilitic and tuberculous affections of the joints. *Syphilitic* arthritis is usually accompanied by other syphilitic symptoms, especially keratitis, and ordinarily yields to antisyphilitic treatment. The differentiation between simple chronic arthritis and *tuberculous* joints is quite difficult, since, as previously mentioned, the latter may follow the former. However, the absence of temperature and failure to obtain a positive tuberculin reaction, speak in favor of chronic nontuberculous arthritis. The finding of a tuberculous exudation in the affected joint, of course, is decisive in the diagnosis.

As the prognosis in protracted cases is very bad, active treatment should be begun early and not too rapidly discontinued, in disgust, because of more or less persistent failure to effect a cure. The salicylates with small doses of sodium iodide internally and 50 per cent ichthyol ointment externally should be given a thorough trial. Where stiffness and swelling of the joints prevail, daily gentle massage preceded by a hot local bath and followed by hot moist compresses often works wonders. Passive motion should be practiced early, and where the contractures are very pronounced one should not hesitate to reduce the same under primary anesthesia and proceed with the treatment just outlined. Concomitant acute symptoms should be treated in the same manner as in acute rheumatism, and when there is reason to believe that the diseased condition is the result of faulty metabolism (intestinal intoxication or uric acid diathesis), the dietary should be regulated accordingly (exclusion of meats, acids, liquors, etc.). Hypertrophied tonsils should be promptly enucleated, and decayed teeth, which cannot be filled, removed.

R	Natrii Iodidi	gr. xv	1.00
	Ext. Hyosciami Fl.	m. vi	0.40
	Natrii Salicyl	℥ i	4.00
	Syr. Sarsaparillæ Comp.	℥ i	30.00
	Aq. Destil.	q. s. ad f ℥ iii	90.00
	M.		

S.—One teaspoonful every four hours, for a child four years old.

Still's Disease

This affection generally sets in during the first three or four years of life and attacks girls more frequently than boys. It is characterized by gradually developing stiffness and enlargement of several joints, beginning with the knee, wrists and cervical vertebræ, and gradually extending to the fingers and toes. It differs pathologically from rheumatoid arthritis or tuberculosis in that it is free from destructive or proliferating processes of the bony structures. As may be readily determined by the roentgen ray examination, the enlargement of the joints is due purely to thickening of the soft tissues. Aside from the articular involvement, Still's disease is characterized by a more or less marked enlargement of the lymphatic glands (axillary, cervical and mesenteric) and of the liver and spleen. It is occasionally associated with a slight rise of temperature, and shows a tendency to pericardial and pleural affections.

It is a very chronic, incurable affection of unknown etiology. Its progress may be partially arrested by the therapeutic measures outlined under ''Chronic Rheumatism''.

Rheumatismus Nodosus Infantilis, Erythema Nodosum, Peliosis Rheumatica (Purpura Rheumatica)

These three distinct diseased conditions are grouped together to facilitate their identification. They have several symptoms in common, and bear a close resemblance to rheumatism. Their true nature, however, is a matter of conjecture, and with our present ignorance as to the identity of the specific rheumatic germ, there are no means of corroboration or of contradiction of any of the numerous assumptions advanced by different authorities.

Rheumatismus Nodosus Infantilis

This disease is peculiar to early childhood and occasionally follows a protracted or recurrent attack of rheumatism, especially in association with grave cardiac manifestations. It is characterized by the (often symmetrical) appearance, chiefly about the joints and the tendon insertions, of several nodules (*noduli* or *osteomata rheumatici*) which grow to a perceptible size, and then either undergo regressive, fatty metamorphosis and absorption, or persist, become calcified and acquire a bony consistence. The nodules (*exotoses*) vary in size from a small pea to a plum and in number from one to a hundred. They are at first soft, flat and painful or tender to the touch, and later they become harder and rounder, resembling the fibromatous and osteo-

Fig. 94

Fig. 95

Fig. 96

Still's Disease in a boy five years old. (G. R. Pisek.)

Fig. 94.—Showing the arthritis being multiple.

Fig. 95.—Periarticular changes in the left wrist joint.

Fig. 96.—Symmetrical changes in the periarticular soft parts of the knees and ankles.

matous growths observed in "Myositis Ossificans" and in "Multiple Exostoses" (*q. v.*).

Treatment.—Antirheumatic.

Erythema Nodosum

Until recently this affection has been looked upon as a skin disease pure and simple. The sudden appearance, the rise of temperature, the self-limited course, and its association with more or less marked constitutional symptoms and occasionally grave complications (principally rheumatic pain, bleeding from mucous membranes and heart trouble), stamp it, however, as an acute infectious disease of obscure etiology. Locally, it is characterized by the appearance, chiefly on the anterior portion of the lower legs and forearms, of from a pea- to a walnut-sized, pale red, painful nodules which at first resemble contusions (erythema contusiforme). They gradually disappear, changing in color to bluish, green and yellow within from two to three weeks, as a rule, without any specific medication.

Treatment.—Complications of the heart and joints demand antirheumatic treatment.

Peliosis (Purpura) Rheumatica

(Schönlein's Disease)

The local manifestations of this affection consist of variously sized bright- to bluish-red hemorrhagic spots which are uninfluenced by pressure with the finger. Here and there they present a central papular hardness. The eruption is usually limited to the lower extremities, especially about the knees and ankles, but the upper extremities may be affected as well. The appearance of the eruption is preceded and accompanied by urticaria, articular pain and swelling, occasionally soreness of the soles of the feet, and difficulty in walking. Fever and constitutional symptoms are ordinarily slight. Occasionally we find edema of the face, slight intestinal hemorrhage and enlargement of the spleen. The hemorrhagic spots usually disappear in from ten to fourteen days.

The prognosis is usually favorable, but the disease manifests a tendency to recurrences, and to cardiac complications.

Treatment.—Symptomatic: salicylates; daily intestinal irrigation with a warm bicarbonate of soda solution (one ounce to 1 quart of water); rest in bed. Light diet.

Myositis

(INFLAMMATION OF THE MUSCLES)

The causes of myositis are very numerous. We had occasion to refer to scarlatinal and rheumatic myositis. It may also be traumatic, gonorrheal, syphilitic and tuberculous in nature, and is occasionally observed in connection with other infectious disease, *e. g.*, typhoid. Myositis is characterized by pain, swelling and loss of function of the affected muscles, and, in protracted cases, by contractures. Where pain predominates and the swelling is slight, myositis may readily lead to diagnostic errors, as emphasized in the discussion of "Muscular Rheumatism." (See p. 416.) Traumatic, syphilitic and tuberculous myositides are prone to lead to suppuration, while simple so-called rheumatic myositis eventually subsides either spontaneously or under antirheumatic treatment.

Polymyositis

This form of general myositis is of much graver nature than the aforementioned varieties. It occurs either primarily, without any apparent cause, or secondarily as a result of parasitic infection, such as trichinæ, echinococci, cysticerci, etc.

Preceded by prodromata of a few days' duration, consisting of headache, muscular pain, anorexia, and slight fever, the condition rapidly grows worse; the temperature rises, and *edema* of the eyelids and face appears which soon spreads over the entire body. Beginning also with the face, the entire musculature of the body (least marked in the hands and feet) rapidly becomes stiff, board-like, and very painful, so much so that the different functions of the body (mastication, deglutition, respiration, etc.) are interfered with and the condition greatly resembles that of cerebral rigidity.

In some cases cutaneous edema predominates (*dermatomyositis*), in others a hemorrhagic condition of the skin and mucous membrane (*polymyositis hemorrhagica*). Some cases develop very slowly and lead to overgrowth of the connective tissue (*myositis fibrosa*). In trichiniasis the polymyositis is usually preceded by gastrointestinal disturbance, and the stools and the muscles reveal trichinæ spiralis. The blood shows marked eosinophilia.

In children the course of the disease is usually milder than in adults and, as a rule, ends in recovery.

Treatment.—Symptomatic: thorough cleansing of the alimentary

tract; relief of pain by antispasmodics, and, in *trichiniasis,* large doses (tablespoonful every 3 or 4 hours) of glycerine.

Myositis Ossificans

Myositis ossificans multiplex progressiva is a disease of childhood, the majority of the cases on record having been observed in children under ten years of age. Anatomically, it is characterized by progressive interstitial connective-tissue proliferation, with consecutive ossification. The affection begins with the muscles of the neck and back, then spreads to those of the extremities, and, finally, involves the masseter and temporal muscles.

The etiology of the disease is unknown.

The onset is sudden with fever, and a soft, painful swelling of a section of a muscle, over which the skin appears reddened and edematous.

The febrile symptoms soon abate, but the swelling in the muscle persists, and gradually—it sometimes takes years—assumes a bony consistence. Several muscles may thus become affected, leading to disturbance of motion, rigidity and deformities, and ossification of a large portion of the body so that the patient becomes bedridden for life. The prognosis, therefore, is grave, and life is endangered early if the muscles of mastication and respiration are involved.

Treatment.—Avoidance of traumatism; the salicylates and the iodides internally and externally; gentle massage and hot baths.

Multiple Exostoses

Bone tumors in children may be congenital or acquired. The latter variety has been spoken of in connection with rheumatism. (See p. 422.) Congenital exostosis may escape observation for several years and then erroneously be attributed to acquired causes. The etiology of congenital exostosis is obscure. Some cases are traceable to syphilis hereditaria. Some authors are inclined to attribute it to a disorder of growth. Bone tumors localized in the immediate neighborhood of joints and interfering with motility should be extirpated. Underhill (Jour. Exp. Med., July, 1920) among others suggests that in the early stages of cartilaginous exostosis, during the proliferative cartilage changes, the progress of the disease may possibly be checked by proper dietary procedures, and especially by restriction of calcium and magnesium intake.

Fig. 97.—Multiple exostoses. The tumors, varying in size from a pea to a walnut, were especially numerous at the costosternal articulations, the wrist-, knee- and ankle-joints.

Meningitis Cerebrospinalis,

Poliomyelitis Anterior,

Encephalitis Lethargica,

Parotitis Epidemica

(MUMPS)

Primary, idiopathic, epidemic parotitis is a contagious and infectious affection of the glandular substance (acini and the ducts) and the interstitial tissue of one or both parotid glands. It most frequently attacks children of from two to twelve years of age, more rarely younger and older children. One attack usually confers immunity for life.

Secondary or metastatic parotitis is not rarely met as a complication or sequel of divers infectious diseases and has nothing in com-

mon with epidemic parotitis. Infection occurs through the mouth or throat. The specific microorganism is still unknown.

After an incubation period of from ten to twenty days and a prodromic stage of about forty-eight hours' duration (marked by general malaise, pain in the region of the ear and throat), typical epidemic parotitis is characterized by a gradually increasing swelling of the parotid gland in front and below the ear and along the angle of the lower jaw. The swelling increases up to the third or fourth day, remains stationary for another two or three days, and then rapidly subsides. Quite frequently after subsiding in one parotid the

Fig. 98.—Epidemic mumps.

inflammation passes on to the other; more rarely both parotids are involved simultaneously. The overlying skin is usually colorless; more rarely, pale red, glistening and painful. Exceptionally the glands undergo suppuration (probably due to mixed infection!) or chronic induration. The inflammation may extend to the other salivary glands, or to the lymphatic and lacrimal glands, involving the tonsils, lids, conjunctiva, and less frequently the testicles, or ovaries, vulva or breast—usually on the same side as the affected parotid. Occasionally the submaxillary glands alone are involved, and, where the parotitis is bilateral and severe, there may be a confluence of the bilateral tumors.

Except pain in swallowing, in opening the mouth, chewing, turning the head, etc., headache, occasionally vomiting, and a rise of temperature during the first or second day of the disease, the patient usually suffers no discomfort. Of course, the symptoms are materially changed if the testicles (orchitis parotidea) or ovaries, etc., are involved, or if complications make their appearance—rather rarely to be observed in cases of ordinary severity. Otitis and nephritis form the most frequent complications. They may occur during convalescence, less often during the acme of the disease. The nephritis is usually hemorrhagic, but benign, in nature. The otitis not rarely leads to temporary deafness. Other complications of parotitis on record are: meningitis, encephalitis, divers paralyses, psychoses, pericarditis, endocarditis, arthritis, etc.—the same as are apt to be met in many other acute contagious and infectious diseases. Notwithstanding the possibility of grave complications and sequelæ, the prognosis of parotitis is almost always favorable, rarely calling for any elaborate therapeutic measures.

Treatment.—A few days' rest in bed, fluid diet, the salicylates for the relief of pain, and local application of lead or potassium iodide ointment with or without 10 per cent of ichthyol, or oil of hyoscyamus, covered with absorbent cotton, usually suffice to effect a cure in the majority of uncomplicated cases. Complications should be treated according to indications. Surgery should not be resorted to unless there be definite signs of suppuration. It is advisable to isolate the patient for about three weeks.

Parotitis may be mistaken for swellings in the same region resulting from stomatitis, alveolar periostitis, retropharyngeal abscess, and infected glands from other causes. Bearing in mind the cause, consistency and location of the tumor, the presence or absence of an epidemic, and the course and duration of the disease, there ought not to be any great difficulty in arriving at a correct diagnosis.

The course of secondary parotitis differs with its cause.

Pertussis

(Tussis Convulsiva, Whooping-cough)

Whooping-cough is a highly communicable epidemic and sporadic affection, during its height characterized by sudden more or less frequent paroxysms of coughing which are from time to time interrupted by deep, stridulous inspiration and followed by a period of apparent euphoria of variable duration. The specific germ of the disease is still unknown, although there seems ample reason for the belief that

the bacillus described by Jochmann, Krause, Bordet and Gengou is the immediate cause of the disease.

As a rule, the course of pertussis is divisible in three distinct stages: stadium catarrhale, convulsivum and decrementi.

The *stadium catarrhale,* which lasts about ten days, begins after an incubation period of from five to nineteen days. It is sometimes preceded by a few indefinite prodromata, consisting of loss of appetite, languor, restless sleep, and slight fever; and as these symptoms gradually disappear they become replaced by those of a simple catarrh of the upper air passages, so that the advent of the grip or measles is often suspected. At first the cough is short, hacking, sometimes croupy in character, but it steadily grows worse, although returning at longer intervals. It is especially troublesome at night, and what, as a rule is particularly characteristic of the whooping-cough, the cough fails to respond to the remedies usually efficient in ordinary "coughs and colds." Toward the end of the catarrhal stage the child is off and on attacked by a paroxysmal loose cough, thus indicating the early advent of the second, convulsive stage, of the affection.

The *stadium convulsivum* may last from two to four weeks or, if let run at random, as many months. The cough is violent and explosive, each paroxysm being often preceded by a slight aura, by vomiting, sneezing, etc., so that older children are usually aware of its approach.

Children able to walk usually run towards a person or object to support themselves during the attack, and infants manifest the approach of the paroxysm by a sudden outburst of crying. Each paroxysm which lasts from one-half to five minutes consists of a number of short, barking, expiratory acts of coughing, from time to time interrupted by deep whistling or stridulous inspirations—which constitute the "crow" or "whoop"—and is ordinarily (sometimes folfowed by a second or third fit of coughing) concluded with the expulsion of a glassy, tenacious mucus and often also vomiting of food residue. During a paroxysm the face is at first red, then cyanosed and the veins in the neck swell. As the attacks grow worse, there is considerable venous stasis, puffiness of the face (which remains occasionally permanent) especially at the eyelids; there may be bleeding from the nose and throat, in the skin, conjunctiva, more rarely from the ear (rupture of the drum membrane, which heals spontaneously), in the meninges, etc. In delicate and younger children a paroxysm is not rarely associated with involuntary defecation and urination, and at times also with general convulsions. The number of paroxysms varies between ten and sixty in twenty-four hours. They

are more frequent when the patient lives in unhygienic surroundings, after overloading the stomach, on excitement from any cause (crying, laughing, etc.), irritation of the nasopharynx and larynx, etc. (often a useful means of diagnosis!). In mild and moderately severe cases the child is apparently quite well between the attacks; in very severe cases, however, the patient is weak, pale, emaciated and suffering from troublesome bronchitis and often from a number of the other grave complications soon to be related. Under proper treatment the paroxysms in uncomplicated cases are, as a rule, more or less checked after from ten to twenty days. The paroxysmal stage is then followed by the regressive stage, *stadium decrementi*. The attacks become less frequent, they lose their typical character, the cough returns to the original catarrhal type and finally abates entirely. This declining stage ordinarily lasts from two to three weeks. Occasionally, however, especially in cases exposed to unsanitary conditions and careless treatment, this stage may continue for months and be interrupted by relapses which often undermine the patient's constitution and lead to irreparable lesions in different organs of the body.

Divers *complications* and sequelæ have been noted: *Of the lungs:* capillary bronchitis, bronchopneumonia, emphysema, and bronchiectasis, phthisis and acute miliary tuberculosis (as a result of caseation of the bronchial glands) ; *of the heart:* dilatation, pericarditis, and myocarditis; *of the brain:* divers paralyses (hemiplegia, facial, laryngeal, etc.), hemorrhagic or tuberculous meningitis, encephalitis, softening of the brain, mental affections, such as imbecility, idiocy and different forms of insanity; *of the spinal cord:* myelitis, hemorrhagic inflammations, and polyneuritis; *of the ears:* otitis, with or without permanent deafness; *of the eyes:* amblyopia, amaurosis; also nephritis, sublingual ulceration (as a result of friction of the sublingual tissues against the teeth during a paroxysm) severe epistaxis, and emphysema cutis (pneumohypoderma, *q. v.*) from rupture of some pulmonary alveoli. Delicate, especially bottle-fed babies, not rarely suffer from gastroenteritis with subsequent marasmus. Finally, sudden collapse from respiratory and heart failure may ensue at the acme of a protracted fit of coughing.

Fortunately, the cases are not all of so grave a nature and so dreadful in their consequences. Numerous abortive cases are on record in which the second stage is devoid of the "whoop" (sometimes replaced by attacks of sneezing), and the third is of very brief duration, so that in the absence of an epidemic or a definite source of infection there is justification for a doubtful diagnosis. When the whoop is absent, some assistance in the diagnosis may be obtained by a careful

examination of the blood, which will show that during the second stage the polynuclear cells are increased twice in number, and the lymphocytes about four times. Of diagnostic importance is also the fact that the urine has a high specific gravity (1,022-32) and contains an excessive amount of uric acid crystals. The diagnosis is often almost impossible during the first stage of the affection especially if following measles, which is quite frequently the case, and time alone is the only reliable guide.

No other communicable affection of childhood is so lightly regarded by the laity and so carelessly treated by the physician as that under discussion. Notwithstanding the fact that it prevails during the greater part of the year; that its mortality ranges between 4 per cent to 6 per cent as an immediate result of the disease, and at least as high as 10 per cent in consequence of complications and sequelæ,* no strenuous effort is being made to still its ravages, to arrest its spread or to abort its course. The fallacious impression has gained firm ground that whooping-cough "must run its course of from six to eighteen weeks," and even the scientific, practical physician wisely nods his head in affirmation and despair, lest he be ridiculed by the therapeutic nihilist. One has to be bold to venture to claim success in allaying the spasm, reducing the number of paroxysms, and preventing the dreadful complications of the disease; and the one who dares to proclaim the possibility of cutting short the lengthy course, courts everlasting infamy! All the same, the severest attack of whooping-cough properly treated may be rendered almost innocuous, or at least free from grave consequences.

Treatment.—As soon as pertussis is suspected the patient should be isolated, and given pertussis vaccines (3 or 4 doses) as a prophylactic. Immunization should also be resorted to in all the other children coming in contact with the patient. Isolation should be practiced principally

---

*Statistics compiled by Morse from the United States Public Health Reports show that comparative death rates per hundred thousand are as follows:

Whooping-Cough	11.4 per cent.
Scarlet Fever	11.6 per cent.
Measles	12.3 per cent.
Diphtheria	21.4 per cent.

He states, furthermore, that 94.5 per cent of the deaths from whooping-cough in the United States occur in children under five years of age, as follows:

Under one year of age	57 per cent.
In the second year	23 per cent.
In the third year	8 per cent.
In the fourth year	4 per cent.
In the fifth year	2½ per cent.

It can, therefore, be seen that the mortality from whooping-cough is higher in those of tender years—being more than twice as high under one as between one and two; and more than five times higher under two years than between two and five. If, to these statistics, we add many of the reported deaths from bronchopneumonia supervening on whooping-cough, the mortality from the latter would be still larger.

SPECIFIC COMMUNICABLE DISEASES

during the expectorating period—at least three weeks. The sputum should be collected in tissue paper or gauze and immediately destroyed.

Fresh air being the most essential and efficient therapeutic measure, the child should, except in the presence of grave complications, be kept outdoors the greater part of the day, and the rooms constantly aired with the patient indoors. Whenever possible, two or more rooms should be made use of. The food should be bland and strengthening, and given in small amounts, preferably after the paroxysms. The clothing should correspond with the season of the year. We possess no ideal specific against the disease, yet pertussis vaccine* (in gradually increasing doses from 500,000,000 to 2,500,000,000), administered at first daily and later once or twice a week, undoubtedly influences the course of the disease very favorably—even though not specifically. Moreover, a great deal can be done to lessen the number and severity of the paroxysms by resorting to the following medicinal agents:

R   Olei Eucalypti   ℥ ii | 8.00
      Tr. Benzoini Comp.   q. s. ad f ℥ ii | 60.00
      M.
      S.—One teaspoonful in a pint of hot water to be used as an inhalation through a croup kettle three times a day.

R   Quinine Ethyl Carbonate or
      Diquinine Carbonic Ester (Euquinine
            or Aristochin)   ℥ ss | 2.00
      Syr. Simplicis   ℥ ii | 60.00
      M.
      S.—One teaspoonful every two to four hours, according to the severity of the paroxysms, for a child three years old.
      In older children the subsulphate of quinine or quinine tannate may be given instead.

R   Creosoti Carbonatis   ℥ iv | 15.00
      S.—Two drops for every year of the child's age.

I wish to direct particular attention to the marked antispasmodic value of calcium and sodium hypophosphites in mitigating and often completely arresting the "whoop" of pertussis. I have been led to administer these drugs after noting the close resemblance between the manifestations of laryngeal spasm of spasmophilia (spasmus glottidis) and those of whooping cough and observing the beneficent action of lime and soda hypophosphites in spasmophilia. Furthermore, it seemed to me quite plausible to assume that, as is claimed in spasmo-

*It should be used within four months of the date of manufacture. (W. C. Davison, Jour. Am. Med. Assn., Jan. 22, 1921.)

philia (*e. g.*, in tetany), the whoop of pertussis may also be due to some functional disturbance in the parathyroids, arising traumatically as the result of the harassing cough during the earlier stages of pertussis.

These preparations do not in the least interfere with the action of any of the others generally employed, but on the contrary, combine well with them, as for example in the following mixture:

℞ Creosoti Carbonatis	ℨ i	4.00
Syr. Calcii et Natrii Hypophosphitum	℥ i	30.00
Pulv. et Mucilaginis Acaciae	q. s.	
Aquae Anisi	q.s.ad f ℥ ii	60.00
M.		

    S.—One teaspoonful every four hours, for a child three years old.

My results thus far have been very encouraging.

Whenever necessary a small dose of some morphine preparation with or without 2 grains of antipyrin may be administered to induce rest or sleep, and where the heart is weak, a fresh infusion or the tincture of digitalis will prove a grateful addition. Numerous other remedies have been found serviceable, but caution is commended in their promiscuous use.

The paroxysms may frequently be controlled by pulling the lower jaw downward and forward. This manipulation is harmless and painless. Its application is contraindicated only in the presence of food in the mouth or esophagus.

A silk elastic abdominal belt (Kilmer) is useful to allay vomiting and the severity of the paroxysms. Chloroform anesthesia will sometimes relieve the attacks almost magically and should be tried in desperate cases, especially in those associated with convulsive seizures.

Complications and sequelæ arising should be treated according to indications.

℞ Ext. Belladonnæ Fl.	m. iv	0.25
Vini Ipecacuanhæ	m. xvi	1.00
Nat. Bromidi	gr. xvi	1.00
Syr. Amygdal.	ℨ iv	15.00
Aq. Destil.	q. s. ad f ℥ ii	60.00
M.		

    S.—One teaspoonful every two to four hours, for a child two years old.

Whooping Cough in the Newborn

The following remarks are based upon the observation of 16 cases of pertussis in infants of from nine to twenty days old. In all of these

babies the source of infection could be traced to members of the immediate family, although in some of them the positive history was not immediately apparent. For example, in one case the source of infection was traced to a grandfather, sixty-four years old, who for a few weeks had been suffering from a paroxysmal loose cough accompanied by semifainting spells. He had been treated for cardiac asthma. Six infants contracted the disease from their mothers who had been suffering from a protracted cough, supposedly bronchitis, because of the absence of the characteristic whoop. As these infants during the first few days after birth were entirely free from any signs of nasopharyngeal or bronchial catarrh, there is every reason to believe that the infection took place after birth, and, furthermore, that immunity was not conferred upon them by their mothers. In the remaining nine babies the source of infection was readily discerned, since one or more members of the family were afflicted with the disease.

The cases of whooping cough in the newborn thus far recorded are exceptionally few. Among them may be cited the classic cases of Bouchut, Rilliet, Barthez, Currier, Watson, Neurath, and Holt ("Twentieth Century Encyclopedia" and Pfaundler and Schlossmann's "Handbook of Pediatrics"). The meagerness of the literature on the subject, notwithstanding the extremely high mortality which prevails among these cases, tends to emphasize the apparent levity with which pertussis is looked upon even by the profession. Of course, due allowance must be made for the fact that a great many infants succumb to the disease before a correct diagnosis has at all been arrived at. For be it remembered that the symptomatology of pertussis in the newborn differs greatly from that observed in older children. Whereas in the latter, as already stated, we are usually able to distinguish three characteristic stages of the disease, thus, *stadium catarrhale, convulsivum,* and *decrementi,* in the newborn the catarrhal and paroxysmal stages are confluent, while the catarrhal stage is so brief in duration as entirely to escape observation. Beginning with occasional mild sneezing or coughing a few days after birth, it is all at once noticed that the baby is struggling for air with each fit of coughing, turns blue and even black in color, and after a few expulsive efforts of expectoration, followed by gagging and trickling out of frothy mucus from its mouth, the infant falls back pale and exhausted, in semicoma as it were. The paroxysms return at shorter or longer intervals, as a rule, every five to ten minutes. The attacks of apnea are almost invariably associated with temporary arrest of the heart's action, and it is not at all unusual for some delicate infants to succumb

during a paroxysm. I witnessed it in 3 cases—twelve and fifteen days old respectively. Of the remaining cases under my observation 4 recovered, 7 died from bronchopneumonia, or rather hypostatic or passive pulmonary congestion, 1 of cerebral hemorrhage and 1 from inanition. One of the cases of bronchopneumonia was complicated by rupture of the pulmonary alveoli (pneumohypoderma, *q. v.*).

The cerebral hemorrhage complicating pertussis is usually localized, giving rise to mono- or hemiplegia, and when confronted with an infant that has been delivered instrumentally and shows distinct signs of forceps traumatism, the diagnosis is apt to be greatly obscured. In the absence of a positive history of whooping-cough, and more especially in the early stage of the disease, it is often also very difficult to decide, whether or not we are dealing with congenital heart disease or hypertrophy of the thymus gland, since in both these affections more or less marked cyanosis predominates. In the differential diagnosis it is well to bear in mind that in congenital vitia cordis, the cyanosis is either permanent or becomes apparent only during fits of crying. Furthermore, physical examination usually reveals definite signs of heart disease, such as murmurs or pronounced anatomic malformations. An enlarged thymus sufficiently marked to produce grave symptoms usually discloses, on percussion, distinct dulness or flatness over the upper portion of the sternum, particularly to the left as low as the second rib and often also to the back between the scapulæ. Furthermore, the paroxysms of asphyxia in thymus hypertrophy are much less marked and less frequent than in pertussis. Mild cases of whooping-cough may sometimes be mistaken for atelectasis pulmonum, but this condition is usually preceded by asphyxia neonatorum and is not accompanied by sudden attacks of coughing. Some aid in the diagnosis may be derived from a careful blood examination which in pertussis generally shows a pronounced augmentation in the leucocytes, but, as there is always a great relative increase in the lymphocytes in the blood of the newborn, this test is not as decisive in infants as in older children. However, this test may serve to detect the immediate source of the infection and should be applied to the other members of the family who happen to be afflicted with a recalcitrant cough.

Treatment.—In view of the extremely high mortality in pertussis neonatorum our main therapeutic efforts must be directed toward prophylaxis. It devolves upon the obstetrician particularly to guard against transmission of whooping-cough to the newborn, be it by the mother or any other member of the immediate family. Even if there is only a suspicion, the infant must be promptly isolated, and with further

corroborative evidence of the existence of the disease, immediately immunized. Judging from personal observation, the administration of prophylactic pertussis vaccine in full doses is absolutely harmless even in the youngest of infants. If the mother is suffering from whooping-cough, we must stop her nursing of the infant, at least until the infant has been thoroughly immunized. In a number of cases, owing to the frequency and severity of the paroxysms, the infants are totally unable to nurse at the breast, in which event it will be found advantageous to feed them on the breast milk by means of Breck's feeding tube, in small quantities, and at short intervals, in the same manner as practiced with premature babies. The active treatment is very unpromising. In 6 of my cases pertussis vaccine as a therapeutic agent proved useless. Some benefit may be derived from the early administration of bromides and lime and soda hypophosphites (see pp. 433, 434) to arrest the frequency of the spasms; of ipecacuanha, to facilitate expectoration and thus to hasten the termination of the paroxysms, and of strophanthus, to sustain the baby's heart action. The bromides, either potassium or sodium, should be given in sufficiently large doses to induce more or less profound sleep. One grain every three to six hours in the beginning and less frequently thereafter usually answers the purpose. The ipecac, preferably the syrup, should be given in from 3 to 5 minim doses until the cough has thoroughly loosened, and whenever the chest and throat become choked up by the tenacious mucus, it is occasionally of advantage to increase the dose of the ipecac sufficiently to produce emesis. Vomiting, by the way, is Nature's method of relieving the paroxysms of pertussis. The dosage of the tincture of strophanthus should vary with the condition of the infant's heart. Generally, $\frac{1}{2}$ to 1 minim, three times a day, will be found sufficient. Finally, it is most important to remember that an abundance of fresh air is the *sine qua non* in whooping-cough, and that, especially in delicate babies, oxygen by inhalation is worthy of trial.

TUBERCULOSIS

Introductory Remarks

(Prevention of Tuberculosis)

Without denying the possibility of antenatal direct bacillary transmission of tuberculous disease from parents to offspring (six cases of undoubted fetal tuberculosis are on record), it may be set down as absolutely certain that, with but very few exceptions, tuberculosis in in-

fancy and childhood, as in adolescence, is acquired as a result of *post-natal** infection by the tubercle bacillus of Koch. The bacillus invades the human organism principally through the respiratory (by inhalation in about 80 per cent) and alimentary tracts (by ingestion in about 15 per cent, often by swallowing tuberculous material derived from the lungs); less frequently through the skin or mucous membranes (slight traumatism, skin eruptions, etc.), and occasionally also through the blood, when broken down tuberculous foci are carried into the circulation. The readiness with which infection occurs depends chiefly upon the power of resistance of the patient and the environment in which the patient is forced to live. This explains the greater frequency of tuberculous disease in children of tuberculous parentage. An undermined constitution from one cause or another (most particularly acute infectious diseases, such as measles, whooping-cough or influenza) forms an easy prey to the tuberculous germ and, varying with the primary seat of the infection, the natural recuperative strength of the tissues involved, and the therapeutic measures adopted to resist and combat further systemic invasion, tuberculous disease may remain localized or become general, and pursue an acute or chronic course.

The successful management of tuberculosis rests upon a thorough appreciation of the aforementioned facts. We possess no specific remedy against tuberculosis, once fully established, but the disease is certainly preventable and in its incipient stage curable—a great deal more than can be said of a number of nontuberculous, organic affections.

Prevention of tuberculosis in a child must begin immediately after its birth. Every infant should be removed from a tuberculous environment! The air the infant is to breathe should be pure, the room it is kept in sanitary and well ventilated, though warm enough to suit its needs. From earliest infancy the child should be gradually accustomed to outdoor air, and, as he grows older, he should spend most of the day outdoors, except when the weather is particularly bad. In this

---

*In 988 children examined by von Pirquet the reaction was found positive:

0 to 3 months .... 0 per cent	4 to 6 years .... 50 per cent
3 to 6 months .... 5 per cent	6 to 10 years .... 57 per cent
6 to 12 months .... 16 per cent	10 to 14 years .... 68 per cent
1 to 2 years .... 24 per cent	

Bass and Hess, Jour. A. M. A., Jan. 11, 1919, made the following observations:

AGE DISTRIBUTION OF PATIENTS REACTING POSITIVELY TO CUTANEOUS OR INTRACUTANEOUS TEST

AGE	NUMBER TESTED	REACTING POSITIVELY	
		NUMBER	PER CENT
0-6 months	51	4	7.8
6-12 months	38	6	15.8
1-2 years	29	5	17.2
2-3 years	45	14	31.1
3-5 years	28	12	42.8

event he should remain well dressed in front of an open window. Special attention should be paid to his breathing. Any obstruction to free nasal breathing, be it adenoids, hypertrophy of the tonsils, or of the nasal mucous membrane, or deformity of the nasal bones, should be treated or removed without delay. The child should be taught to breathe deeply—few children know how to breathe, as is readily evinced in examining a child's chest. Infants should be encouraged to cry off and on, and older children to recite and sing in the open air. As the child
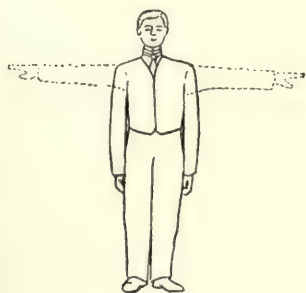
Fig. 99.　　　　Fig. 100.　　　　Fig. 101.

Fig. 102.　　　　Fig. 103.

Figs. 99-103.—Breathing exercises.

grows old enough intelligently to follow instructions, he should be taught the following breathing exercises:

1. Deep inhalation, while raising the arms to a horizontal position; slow exhalation, bringing the arms down (Fig. 99).

2. Deep inhalation with the arms placed lightly upon the front of the lower portion of the chest; slow exhalation, bringing the arms down (Fig. 100).

3. Deep inhalation, while bringing the arms first to a horizontal position, then above the head, and lastly—while still holding the breath—

bending the upper body backward; slow exhalation, while lowering the arms sideways (Fig. 101).

4. Deep inhalation, while bringing the hands together in front of the abdomen, and from here slowly along the thorax and chin above the head and as far as possible back of it; slow exhalation, bringing the hands down to the original position (Fig. 102).

5. Deep inhalation, while bending the upper body as far back as possible, with the hands fixed on the hips; slow exhalation, while resuming original position (Fig. 103).

During the breathing exercises the child assumes the position of military "attention." He breathes with the mouth closed, occupying about four seconds for inhalation, four seconds for retention of the air and three seconds for exhalation. The exercises should be practiced either outdoors or in front of an open window; at first four or five times a day, but, after the child gets accustomed to expand his chest properly during the respiratory act, only once or twice a day or not at all. The breathing exercises, like any other physical work, should not be overdone, and never continued too long as to become tiring. As prolonged holding of the breath interferes with the normal heart's action, it is contraindicated in organic heart disease. Short-distance running, and peaceful outdoor games (hand-ball, basket-ball, and tennis) also are helpful to expand the lungs. The principal benefit derived from these breathing exercises is the purification of the lung tissue by the free inflow and uniform distribution of oxygen, thus preventing pulmonary congestion which acts as a predisposing cause of tuberculous infection.

What pure air does for the prevention of pulmonary tuberculosis, suitable feeding from birth will do for the prevention of tuberculosis of the alimentary tract. It is highly essential ever to bear in mind that tubercle bacilli rarely, if ever, survive the action of normal digestive juices. The gastroenteric tract, especially the stomach, therefore, should be spared pathologic alteration. Breast milk of a healthy mother or wet-nurse should at all times be the food of choice for an infant up to nine months old. With increasing age the dietary should undergo a gradual change, always selecting, however, such articles of food as will best accomplish the object in view, i. e., ample nutrition for the growth and development of the child with least possible injury to the digestive organs. Overfeeding especially is to be avoided. It goes without saying that contaminated food should never form a part of the dietary. Cow's milk of doubtful purity should be sterilized, and other articles of food of such character boiled. The teeth should receive special attention, since cavities of decayed teeth not rarely harbor tubercle bacilli and

early loss of the permanent teeth forms one of the principal causes of acute and chronic dyspepsia—as a result of insufficient mastication of the food—and indirectly enhances the development of tuberculosis. Children should be taught to eat slowly, and to refrain from eating between meals.

Tuberculous infection through the skin and contiguous mucous membranes should be guarded against by scrupulous cleanliness of these structures, avoidance of external injury and skin eruptions, and by immediate treatment of open wounds and all such skin lesions as are associated with itching and compel scratching. Those intrusted with the care of babies and older children should be instructed to give their charges a tub bath in the evening and a sponge bath in the morning followed by gentle rubbing of the entire body. Of course, the bathing should include careful cleansing of the nails, which should be kept clipped short, of the ears, of the nose and scalp, and, in older children, also of the teeth. From earliest infancy children should very gradually get accustomed to cool sponge baths. At first the infant may be given a cool alcohol sponge bath, and after toleration has been established the alcohol should gradually be replaced by water, and finally by full cool tub or shower baths (except during cold winter months). The advisability of cleansing the infant's mouth is still a matter of opinion. I am inclined to favor gentle *wiping* of the infant's mouth twice daily with a cotton swab dipped in sterile water. Older children should be taught the use of a soft brush for the teeth and an antiseptic gargle for the mouth and throat. The importance of early removal of nasopharyngeal obstruction to breathing has already been alluded to. This question cannot too strongly be emphasized, for the adenoid tissue, in addition to interfering with free respiration, is surely one of the most rampant sources of tuberculous infection. Skin eruptions should at once be combated. This refers especially to running sores from whatever cause, and to all skin diseases which sooner or later lead to maceration and denudation of the skin. Intertrigo in infants is best prevented by frequent changing of the diapers and keeping the buttocks perfectly clean and dry. The child should be kept from scratching the affected portions of the skin by immediate application of antipruritic drugs and by restraining the child's hands by means of one of the many useful contrivances. Open wounds should be dressed antiseptically until healed. Vaccination wounds especially should receive careful attention. Certain, though it be, that latent tuberculosis is occasionally lighted up through vacinnation, and that tuberculosis has in very exceptional instances been traced to vaccine infected by tubercle bacilli,

it is absolutely settled that the great majority of cases of tuberculosis following vaccination are the result of direct bacillary infection through an unprotected vaccination wound.

Effective as these local measures are in the prevention of tuberculosis, their efficiency is very insignificant as compared with the natural defensive resources of a healthy constitution. Our aim, therefore, should be directed chiefly, from earliest infancy, to rendering the patient, so to say, immune against tuberculosis. This is best accomplished by outdoor life, wholesome nutrition, and sanitary environment. Those showing a tendency to remain delicate in health should reside in the country.

Miliary Tuberculosis

(HASTY CONSUMPTION)

This disease is characterized by wide distribution of the tuberculous lesions. The latter are from a pinhead to a millet seed in size, gray
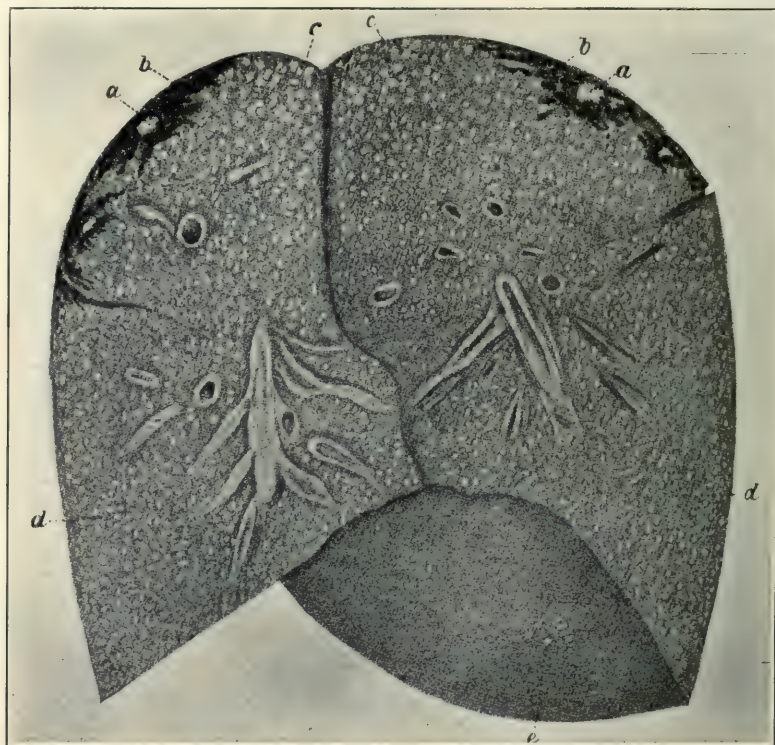
Fig. 104.—Acute pulmonary miliary tuberculosis (cut surface of the lung). *a.* So-called obsolete tubercle (old encapsulated caseous focus). *b.* Induration. *c.* Caseous, partly agminated nodules (transverse section of caseous bronchi). *d.* Submiliary noncaseated tubercle in the true lung tissue. *e.* Tubercle of the pulmonary pleura. One-half natural size. (Langerhans and Brooks, F. A. Davis Co.)

or yellow in color, and firm in consistence. They are found scattered throughout almost all the organs and tissues of the body, but especially the lungs and bronchial glands, intestines and mesenteric glands, the liver, spleen, kidneys and bladder, and the brain and its coverings. They may remain latent for some time, or give rise to indefinite symptoms, such as anorexia, dyspepsia, gastroenteritis, and emaciation, or symptoms of pulmonary phthisis. The outbreak is often determined by some intercurrent disease or traumatism, but once established it usually runs a very violent course.

Fig. 105.—Miliary tuberculosis of the lungs in a child nine years old.

The temperature rises, is intermittent, hectic in character, only rarely drops to normal, and may be associated with chills and sweats. In the beginning, especially in the absence of marked pulmonary symptoms, and in the presence of a large liver or spleen, or both, the disease greatly resembles malarial fever or typhoid. Careful examination, however, reveals the absence of the malarial or typhoidal germs in the blood. Where signs of pulmonary disease predominate, it is readily confounded with lobular pneumonia. In such cases the diagnosis is extremely difficult and often can be decided only by microscopic examination of the

sputum (frequently negative) and the tuberculin test. As the disease advances the diagnosis may be based upon the extreme emaciation, multifariousness of the symptomatology, the violence and persistence of the febrile attacks and x-ray examination.

The symptoms and course of the disease differ with the seat of the lesions. The lungs almost invariably show signs of consolidation (dulness, crepitant râles, dyspnea, cyanosis, short cough), and the intestines rarely escape involvement. In some cases brain symptoms (apathy, jactitations, stupor, localized convulsions, tubercles in the choroid, etc., up to a typical picture of meningitis) predominate; in others again, symptoms of disturbed circulation (marked cyanosis, edema, rapid feeble pulse, anemia and exhaustion, etc.) prevail. The latter phenomena usually precede the fatal issue, which generally occurs within from four to eight weeks. Cases running a subacute course may last a few weeks or months longer, are not rarely erroneously diagnosed and treated as marasmus, their true nature not being detected until postmortem. It is in those cases, particularly, that the von Pirquet or intracutaneous reactions and a roentgenogram are so helpful in the diagnosis, and should always be resorted to early. Then and then only may our efforts to arrest or possibly cure the disease prove successful.

For details of treatment, see p. 449.

Phthisis Pulmonum

(Tuberculosis of the Lungs and Bronchial Glands)

The lungs proper, the bronchial glands, or both, may be the primary seat of tuberculous deposits. The upper lobes are more frequently affected than the lower, and the portions adjacent to the bronchial glands more so than the remaining parts. The pathologic changes consist essentially in the formation of variously sized caseous nodules filled with colonies of tubercle bacilli and large so-called giant cells, and subsequent softening and breaking down of the nodules, forming cavities which may vary in size from a pea to a walnut, or larger. There is usually an endarthritis with miliary tubercles in the walls of the blood vessels. The pleura is thickened and the lining of the larynx, trachea and bronchi ulcerated. In some cases, especially in those receiving early and suitable treatment, the tuberculous process is arrested by encapsulation of the necrosed structures by newly formed connective tissue, leading to contraction and formation of a firm cicatrix. In this event the enclosed masses are in part absorbed, and in part calcified. The tuberculous affection of the bronchial glands also consists in hyperplasia and caseous degeneration. This process usually (sooner or later) ex-

Fig. 106.—Tuberculosis. Horizontal section through the tuberculous lower lobe of the right lung of a two-year-old child. (*a*) caseous focus in the region of the anterior border; (*b*) nontuberculous posterior border; (*c*) transverse section of bronchus; (*d, d¹*) caseated lymph glands; (*e*) pulmonary vein; (*f*) point of adhesion of the *vein e* with the *lymph gland d¹*; (*g*) tubercle in the lymph vessels of the lung parenchyma; (*h*) periarterial, (*i*) peribronchial, (*k*) perivenous tubercles; (*l*) lymph-vessel tubercles of the pleura; (*m*) tubercle in the connective tissue of the hilus of the lung. ✕ 3. (Ziegler.)

tends to the contiguous structures, exerts pressure upon the adjacent blood vessels, nerves, and bronchi, and, after forming adhesions, may displace, erode and perforate these parts. In this manner not only may tuberculous infection be rapidly carried throughout the lungs and more distant organs (producing an acute or chronic tuberculous pneumonia), but perforation of a blood vessel or bronchus, or entrance of caseous masses into the trachea may unexpectedly produce sudden and often fatal hemorrhage or suffocation.

The symptoms vary with the primary seat of the lesion and the subsequent pathologic changes. A small tuberculous focus, be it in the lung or bronchial glands,* rarely gives rise to any definite clinical phenomena. As a rule, in the beginning the disease pursues a latent course. This is especially true in infants. The child is pale, loses in weight, often notwithstanding good appetite, gets tired on the slightest exertion, "hems" and coughs a little, and the temperature rises somewhat in the evening. Sooner or later the symptoms become more distinct. Emaciation, cough, and gastrointestinal disturbances increase in severity; the child suffers from dyspnea, and, if the bronchial glands are involved, from paroxysmal attacks of coughing, greatly resembling pertussis (the cough has a metallic timber with a characteristic expiratory whoop). This cough is the result of pressure exerted by the enlarged bronchial glands upon the pneumogastric and recurrent nerves. Physical signs, however, are often still wanting. Occasionally, percussion over the mediastinum may reveal increased dulness, but in infants this symptom is not pathognomonic in view of the physiologically large thymus. Indeed, the disease is often not detected until grave, not rarely fatal, symptoms (e. g., hemoptysis, hectic fever) announce the seriousness of the condition. The diagnosis of pulmonary phthisis in infants, therefore, must be based upon the entire clinical picture, rather than the local symptoms. If, for example, bronchial catarrh is associated with progressive emaciation, multiple glandular swellings, protracted diarrhea and possibly also some bone or joint disease, the diagnosis of tuberculosis is justifiable, even though careful examination of the thorax fails to reveal pulmonary consolidation or cavity. For corroborative evidence we should carefully examine the child's sputum (obtained by means of a catheter introduced to the base of the tongue) for tubercle bacilli, and employ the tuberculin and complement-fixation tests and the Roentgen-ray.

In older children the symptomatology of pulmonary tuberculosis is essentially the same as in adults. Its onset is usually insidious, and

---

*According to D'Espine, bronchophony heard along the spinal column below the second or third dorsal vertebra points strongly towards enlargement of the bronchial lymph nodes.

quite frequently follows delayed convalescence from some acute disease, such as pertussis, morbilli, broncho- or lobar pneumonia, and the like. The child fails fully to recuperate, is pale, thin, and feeble, suffers from slight shortness of breath, dry cough, chilliness and fever. At first these symptoms are more or less masked, but as the lung destruction advances the symptoms and physical signs grow rapidly worse. The cough becomes persistent, often distressing, especially at night, and attended by expectoration and pain. The fever is intermittent or remittent (hectic) in character. It is usually normal or slightly above normal in the morning, and from two to three degrees higher in the evening. It is often preceded by chilliness and followed by profuse sweating. During the height of the fever the cheeks are usually brightly flushed and

Fig. 107.—Phthisis pulmonum in a child twenty months old.

contrast strongly with the remaining portions of the face which are deathly pale. Night sweats are often observed early in the course of the disease. With further progress of the disease the expectoration becomes mucopurulent or purulent, nummular, and streaked with blood; the fever more irregular, and attended by great exhaustion, and the emaciation profound.

The agony may be further aggravated by the concurrence of a number of painful complications. The disease may extend to the pleura (pleuritis sicca or with serous or hemorrhagic effusion); to the trachea and larynx (dysphagia, frequent hemorrhages, and aphonia); to the alimentary tract (colliquative diarrhea); and where the bronchial glands or pleura are involved, to the pericardium (pericarditis). By

this time, and sometimes at an earlier period the child presents a characteristic, ghastly appearance. The cheeks are hollow, the eyes and temples sunken, the bones of the face and the ears prominent, the nose is pointed and drawn, and the hair thinned, lusterless and brittle. The face is either deathly pale or marked by florid redness along the zygomatic regions. The neck is wasted, the supra- and subclavicular spaces are depressed, the shoulders stoop, and the shoulder blades project wing-like far beyond the shrunken, immovable spine. The thorax is narrow and contracted, and the ribs overlap each other, effacing the intercostal spaces. The abdomen is flat or deeply sunken below the strikingly prominent pelvic bones. The extremities are mere skin and bone and their epiphyseal ends seem greatly enlarged as they protrude through the wasted, arid integument.

The physical signs vary with the stage, location and extent of the lesions. As already mentioned tuberculosis of the bronchial glands may by physical examination entirely escape observation. The same holds true of cases where the tubercles are scattered throughout the lungs and do not coalesce. On the other hand, where pulmonary consolidation (tuberculous pneumonia) occurs early and progresses rapidly, the physical signs resemble those of ordinary pneumonia, i. e., dulness on percussion, prolonged expiration, increased vocal fremitus, fine, coarse and crepitant râles, and bronchial breathing. To these may be added the physical signs of dry or serohemorrhagic pleurisy (see p. 330), which frequently accompanies phthisis pulmonalis. Where cavities are formed, the physical signs consist of cavernous respiration, bronchophony or pectoriloquy. The percussion resonance is amphoric, if the walls around the cavity are thin and tense; cracked-pot sound, if the walls are thin and relaxed, and dull, if the walls are thick. If pneumothorax is present, the percussion sound in tympanitic, and the respiratory murmur is lost; while hydropneumothorax gives rise to tympanitic resonance above the water line, dulness below, and metallic tinkling on auscultation.

The poignancy of the clinical picture just depicted notwithstanding, errors of diagnosis are quite possible. Pulmonary phthisis may readily be confounded with bronchial dilatation, localized empyema, fetid bronchitis, pulmonary gangrene, and syphilis. In view of the prognostic importance of an early diagnosis of tuberculosis, it is imperative to employ every means of diagnosis (especially repeated examination of the sputum, the tuberculin reaction and the x-ray) to clear up all doubt.

The course and duration of phthisis pulmonum ranges within very wide limits. Not only is it true that tuberculosis may proceed a latent

course for months or years and suddenly break out—often after some trivial cause, such as vaccination, measles, etc.—and rapidly end fatally under symptoms of lobular or lobar pneumonia and the like, but postmorten examinations have repeatedly established the fact that after existing for some time, with or without indications of their presence, tuberculous lesions may heal spontaneously never to return. As a rule, however, pulmonary phthisis in young children runs quite an acute course. Unless the disease is arrested in its incipiency, infants usually succumb to it within from four to eight weeks either from the immediate effects of the pulmonary lesions, or as a result of generalized tuberculosis not rarely of the miliary variety. In older children the disease pursues a less violent course, and, as in adults, shows a tendency to remain localized at its originally infected focus until a very late stage of the disease. If the tuberculous process is allowed to continue, death invariably occurs in from two to three years or earlier—either from asthenia (with symptoms of gradual exhaustion, profound anemia, dropsy, etc.) or from apnea (suffocation by sudden hemorrhage, rupture of large cavity, pulmonary edema, etc.). On the other hand, if the tuberculous process is detected in its *incipiency*—which is quite possible with the existing modern diagnostic methods, more especially the tuberculin tests and x-ray—and immediately and energetically treated, the chances for arrest and eventual cure of consumption of the lungs are very good indeed.

Treatment.—The treatment comprises outdoor life, good food, personal hygiene, and symptomatic medication. Whenever possible, tuberculous children should be sent to the country regions where the climate is dry and of equable temperature, so as to allow the patients to enjoy outdoor air the greater part of the day. The climates of New Mexico, Arizona, and Egypt are best suited for the purpose, although a great many patients will be found to do well in Colorado, in the Adirondacks and Sullivan County of New York, in Montana, Wyoming and North Carolina. Those financially incapacitated to take advantage of these climates should be removed to climatically less favorable mountain regions or even to ordinary city suburbs, but at all events should not be left to perish in overcrowded, unsanitary tenement districts. It is often of great advantage to place the child in an up-to-date sanatorium (if possible in a private room) since prophylactic and active therapeutic measures are more accurately enforced (and with less resistance on the part of the patient) under the supervision of a reliable physician and nurse of a properly con-
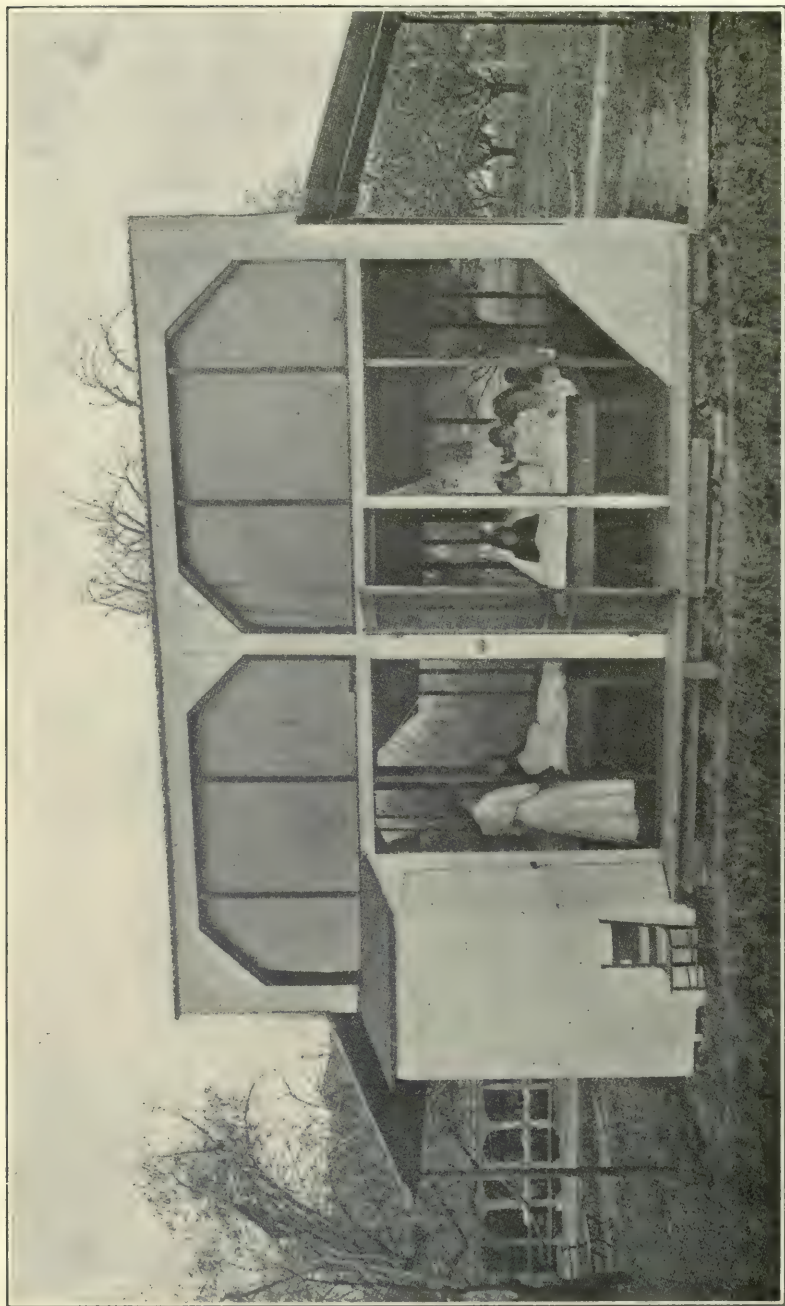
Fig. 108.—A group of tuberculous patients in the outdoor "shack" of the Hospital for Crippled and Deformed Children, New York.

ducted sanatorium, than at the patient's residence among his timid and sympathetic immediate relatives.

The diet should vary with the age of the patient, but should be highly nutritious and liberal. Milk, meat, eggs, fresh fish, oatmeal, peas, beans and lentils, carrots, spinach, asparagus, potatoes, etc., in addition to an ample supply of bread and butter, should form the principal components of the regular meals. Between meals the child should receive plenty of fresh fruit or fruit juices, and, to satisfy its craving for condiments, a moderate supply of sugar, sweet or milk-chocolate or calf's foot jelly, and the like.

The room occupied by the patient should be large and airy, and its windows open day and night, irrespective of season or weather. The child should sleep alone. In addition to a warm cleansing soap bath once a week, it should receive a cool sponge bath twice a day followed by brisk rubbing of the entire body. The underwear should be of thin silk or wool, and the outer garments should vary with the season of the year—always sufficient to keep the patient comfortably warm. In the absence of fever or circulatory disturbance, light exercise that does not fatigue acts very beneficially. Horseback riding is highly to be recommended.

The value of drugs as auxiliaries in the successful management of pulmonary tuberculosis should not be underestimated. It is not very long ago that creosote was almost universally hailed as the specific against consumption. And, while its curative claims had been (as is always being done with new methods of treatment) grossly exaggerated,* its efficiency to relieve distressing symptoms (useless cough) and to aid in arresting the further spread of the tuberculous lesion cannot wholly be denied. Creosote should be given in small gradually increased doses, well diluted in milk, malt extract or red wine. The hypophosphites also are deserving of trial, and may advantageously be combined with malt and cod liver oil, as follows:

℞	Olei Morrhuæ	℥ iv	120.00
	Extr. Malti	℥ i	30.00
	Syr. Hypophosph. Comp.	℥ i	30.00
	Glycerini	ℨ iv	15.00
	Pulveris Acaciæ	ℨ iv	15.00
	Aq. Cinnamomi	q.s. ad f ℥ viii	240.00
	M.		

S.—Two teaspoonfuls three times a day.

*Most recently Chaulmoogra oil has been brought to the front as an efficient antituberculosis remedy (Editorial Jour. Am. Med. Assn., lxxiv, No. 23, 1920.)

The bowels should be kept open, and the appetite improved by means of bitter tonics, especially nux vomica and the compound tincture of cinchona.

In incipient phthisis it is very rarely necessary to resort to opiates or its derivatives to check the cough, but when the latter is distressing, especially at night, those remedies should be cautiously administered as often as indicated.

The management of advanced cases of tuberculosis of the lungs is essentially the same as in incipient cases, except that one is often called upon to arrest hemoptysis (ice bag to chest, morphine hypodermically),* and to check hyperidrosis (sponging of the body with a strong alum solution, atropine by mouth or hypodermically), and to strengthen the heart's action (digitalis and strychnine). In the presence of the aforementioned complications, however, very few children survive—do what you will. Like the flickering flame of the

Fig. 109.—Tuberculosis of the brain (boy four years old). During the protracted course of the disease a marked hypertrichosis developed over the entire body, especially the legs.

candle end, after many ups and downs, slowly but surely, life is extinguished—often at a time when the patient seems on the mend.

Tuberculosis of the Brain

Brain tuberculosis occurs in children (1) as a partial manifestation of general tuberculosis, (2) as tuberculous meningitis, and (3) as brain tumors. The brain lesions are essentially the same in the three clinical types of the disease. They consist in the deposit of tubercles in the brain substance which vary in size from a millet seed to that of a hen's egg. In tuberculous meningitis we find, in addition, inflammation of the pia mater of the brain and sometimes also of the cord and transudation into the ventricles (*chronic hydrocephalus*). The tubercles are usually located in the gray matter—in the large ganglia,

*Artificial pneumothorax may be resorted to in older children.

in the pons and in the cerebellum—and occasionally also in the white
substance. During life, however, it is extremely difficult to determine
the seat of the lesion, except when the latter is large enough to exert
pressure on the vital structures which in their turn give rise to focal
symptoms—as for example, paralysis of the cranial nerves in the dis-
ease of the pons. In the absence of such symptoms tuberculosis of the
brain may exist for months without being detected. This is especially
true of brain tuberculosis associated with tuberculosis of other organs.
As the disease progresses, the symptomatology becomes clearer. The
child suffers from intense headache, convulsions, paresis or paralysis of
some of the cranial nerves or extremities, but even then it is often
only a matter of conjecture whether these pressure symptoms are due
to tubercle or to other tumors. (See ''Tumors of the Brain,'' p. 645.)
The diagnosis is least difficult when tuberculosis of the brain is mani-
fested by meningitis. (See p. 614.) Here lumbar puncture and the
complement-fixation reaction (q. v.) often help to clear up the diagnosis.
Recourse should be had also to the tuberculin test, examination of the
sputum for tubercle bacilli, and ophthalmoscopic inspection of the eyes
for choroidal tubercules.

Tuberculosis of the Abdominal Organs

Aside from the intestinal tract and peritoneum, the spleen, liver,
pancreas, diaphragm, omentum, suprarenals, and the urogenital sys-
tem may also be the seat of tuberculous disease. Except in the rare
instances of invasion of the abdominal organs by tubercle bacilli
through the general circulation (blood or lymph channels), the ab-
dominal organs usually become involved secondarily to intestinal or
peritoneal tuberculosis. As a rule, these latter structures become
infected primarily by swallowing of food, sputum or necrotic tissue
from the nasopharynx contaminated by tubercle bacilli.

Tuberculous Peritonitis

This condition is the result of dissemination of tubercles over the
peritoneum, omentum, and adjacent structures. The inflammation ex-
cited by their presence gives rise to a serofibrinous or hemorrhagic
exudation with gradual agglutination of the inflamed portions, case-
ation and ulceration. Postmortem examination of cases of long stand-
ing usually reveals involvement of the mesenteric and retroperitoneal
glands, amyloid degeneration of the liver and spleen, tuberculosis of
the lungs, and parenchymatous nephritis.

Tuberculous peritonitis is comparatively rare in children under three years of age, but quite frequent in those over this age. The classical variety of tuberculous peritonitis is the chronic form. Occasionally, however, it may pursue a subacute, or even an acute course with chills, nausea, vomiting, acute abdominal pain, and high fever, simulating acute perforative peritonitis of appendiceal origin. In the majority of instances the disease sets in insidiously with symptoms of dyspepsia, anemia, evening rise of temperature, accelerated respiration and pulse, frequent attacks of colic and more or less pro-

Fig. 110.—Tuberculous peritonitis in a baby fifteen months old; she has fully recovered after laparotomy. The von Pirquet test was negative and the diagnosis was based chiefly upon the differential sign mentioned on page 155.

nounced diarrhea. Very soon the characteristic symptoms of the disease are in full bloom. The abdomen is distended and its wall often glistening and traversed by blue lines, the epigastric veins. The umbilicus is either effaced or protuberant. The extremities are emaciated and contrast strongly with the gradually enlarging abdomen. Some portions of the abdomen are flat on percussion, eliciting the presence of fluid or nodular masses, other portions again are tympan-

itic, denoting that part of the abdominal enlargement is due to intestinal gases.

Palpation sometimes confirms the findings on percussion. Occasionally hard, cord-like, painful masses and thickened omentum or adherent intestinal loops are found, of more rarely large tumors or encapsulated abscesses are detected. The latter if situated near the navel (periumbilical tuberculous abscess) may open and discharge through the navel. The abdominal enlargement may persist, or after disappearance of the fluid content and formation of fibrous adhesions the abdomen may retract, become tray-shaped, and remain so until exitus.

If not arrested by therapeutic measures the disease usually runs a very protracted course—months or even years. Remissions are not rare, but sooner or later the symptoms return, sometimes in acute form; the patient wastes away, is troubled by hectic fever, sweats, diarrhea, hiccough, vomiting, dysuria, anuria, and edema of the lower extremities or general anasarca, until death finally relieves him of his agony. Fatal issue may occur also from intercurrent diseases, such as intestinal perforation, tuberculosis of the meninges or lungs.

On the other hand, the prognosis is not as grave if treatment is instituted early, provided, of course, that the disease is limited to the peritoneum.

Unfortunately in the early stage of the disease, the symptoms are not infrequently masked, and a positive diagnosis cannot be arrived at until the pathognomonic signs of the disease have made their appearance, i. e., abdominal distention, circumscribed dulness, emaciation, diarrhea (diarrhea, emaciation and glandular swelling are often absent), hectic fever, and swelling of the inguinal glands. Even then the peritonitis may be confounded with ascites, accompanying cirrhosis of the liver or valvular heart disease. In such cases the diagnosis may sometimes be settled by the tuberculin tests, by a bacteriologic examination of aspirated abdominal fluid or by inoculation experiments. (See also "Chronic Abdominal Enlargement," p. 151.)

Treatment.—As spontaneous cure is extremely rare and radical cures by laparotomy are quite frequent (about 50 per cent), the latter mode of treatment should be resorted to as soon as practicable. Some authors attribute the curative effect of laparotomy to the admission of atmospheric air to the abdominal cavity, others to hyperemia of the peritoneum produced by the operation in a manner similar to that employed by Bier in the cure of tuberculosis of the extremities. Except abundance of sunshine, sojourn at the seashore or mountains and

plenty of wholesome food—which measures should be employed also in conjunction with an operation—all other medical procedures are only of temporary benefit.

Intestinal Tuberculosis

(TABES MESENTERICA)

According to Hess, the bovine type of tubercle bacilli is responsible for 60 per cent of these cases. The tuberculous lesions are usually found in the lowest portions of the ileum, ileocecal region and colon. It is manifested by a tuberculous infiltration of the solitary follicles and mucosa of the intestines, which gradually undergo softening and caseation and finally break down, leaving behind annular ulcers. Tuberculous inflammation of the large intestine may produce so much swelling as to occlude the intestinal lumen. Sooner or later the inflammation extends to the mesenteric glands and the peritoneum. Occasionally the lungs and other organs become involved.

All these manifestations, however, are observed only at the autopsy. During life the symptoms are very obscure. Palpation may reveal enlarged mesenteric glands deep down in the abdomen, but more frequently owing to meteorism they escape observation, and even if palpable are not invariably tuberculous in nature. If, however, this symptom is associated with enlargement of the other glands of the body, stubborn diarrhea (greenish-gray in color, mixed with mucus, pus, and often blood), emaciation and cachexia, sweats and hectic fever, the diagnosis of intestinal tuberculosis is fairly certain. The diagnosis is rendered positive by the demonstration of tubercle bacilli in the stools. The tuberculin test and examination of the sputum often prove decisive in doubtful cases; and complications, such as perforation of the intestines with consecutive peritonitis, settle the diagnosis beyond a doubt. Indeed, in the majority of instances the diagnosis cannot be made until these complications arise, a period at which therapeutic measures almost invariably fail. At all events the prognosis is extremely grave.

Cases of local tuberculosis detected early and treated energetically (chiefly surgically) may recover.

Tuberculosis of the Genitourinary Tract

Urogenital tuberculosis, especially tuberculosis of the kidneys, is comparatively common in children. It occurs either as a manifestation of general tuberculosis or as an independent disease. In the latter event it almost invariably begins in one kidney, and from

here it spreads to the bladder, and to the other kidney. In the beginning the affection is very apt to be overlooked, but, as the tuberculous process advances, the symptoms (pain in the region of the kidney and ureter, thickening of the ureter—as evinced by palpation with the finger in the rectum or vagina—irritability of the bladder, albuminuria, pyuria, and often hematuria) become sufficiently characteristic as to demand careful repeated, bacteriologic examination of the urine
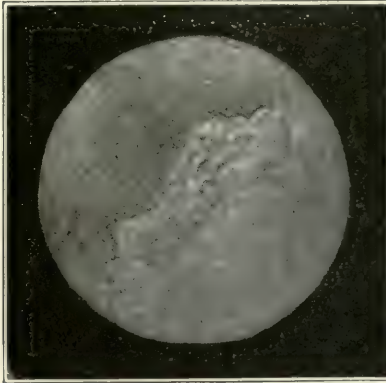
Fig. 111.—Characteristic early tubercular infiltration, as seen through the cystoscope. (Leedham-Green.)
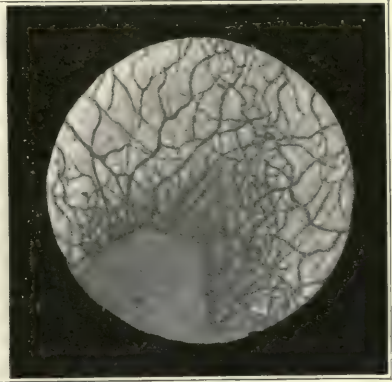
Fig. 112.—A large tubercular ulcer below the orifice of the right ureter. (Leedham-Green.)
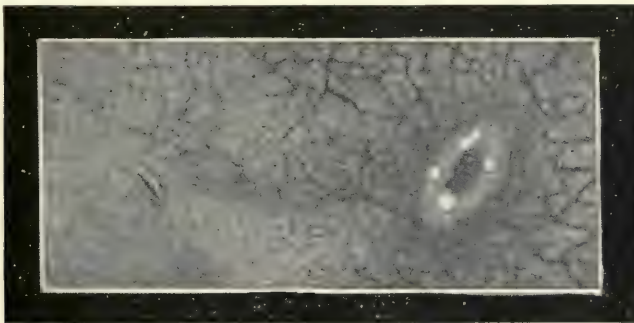
Fig. 113.—Cystoscopic view of the base of the bladder in a case of tuberculosis of the left kidney (Wyatt). The opening of the right ureter is normal; the opening of the left ureter is seen to be gaping, the lips edematous and thickened, showing the presence of small miliary tubercles.

for tubercle bacilli, and cystoscopic inspection of the bladder for tuberculous lesions. Even in the early stage, systematic cystoscopic examination of the bladder will rarely fail to detect tuberculous nodules and ulceration about the opening of one ureter (Fig. 113). In

cases of long standing the lesions are often scattered throughout the
bladder.   As in tuberculosis of the other organs, the tuberculin test
should always be employed to corroborate the diagnosis.   Early recogni-
tion of the condition and prompt surgical treatment are not rarely
followed by permanent recovery.

Scrofulosis

(Tuberculosis of the Skin, Mucous Membranes and Glands)

The tuberculous symptom-complex presently to be described should
not be confounded with similar groups of symptoms which are tran-
sient in character and generally due to strepto- and staphylococcus in-
fection.   Genuine scrofulosis attacks children with undermined consti-
tutions who are poorly fed and cared for, forced to live in damp, dark
and filthy dwellings, and who are exposed to tuberculous infection.
Various skin eruptions, or injuries, exanthemata, decayed teeth, and
diseased tonsils and adenoids, among others, serve as the portals of en-
try to the tubercle bacilli.   The immediate result of the tubercular infec-
tion is hyperplasia, and the more remote effect, caseous degeneration of
the parts primarily involved, and frequently secondary infection of the
neighboring structures.

Clinically, scrofulosis is characterized by simultaneous or successive
involvement of the skin, mucous membranes and lymphatic glands;
chronicity of its course, and a tendency toward slow spontaneous re-
covery, or transition into general tuberculosis.

The skin is the seat of a pustular eruption which resists ordinary
local treatment, generally involves the subcutaneous tissue, and breaks
down, forming slowly discharging abscesses or indolent ulcers.   It is
most frequently situated upon the back and nates, but is found also
upon the scalp and face—probably carried from one part to the other
by scratching by means of infected fingers.

Scrofulosis of the mucous membranes is manifested chiefly by naso-
pharyngitis.   From the nasopharynx the inflammatory process may
spread to the ears, eyes, larynx and oral cavity.

The nasal mucous membrane is red and swollen and discharges a
seropurulent secretion which forms yellowish-green crusts within and
around the nares, producing snuffling respiration and excoriation of
the upper lip.   A similar acrid discharge is usually observed from the
ears (bilateral otorrhea).   Both the nasal and aural discharges may be-
come purulent and fetid; in the first instance, by extension of the inflam-
mation from the nasal mucous membrane to the cartilage, periosteum

and even nasal bones (sometimes marked nasal deformity) ; in the second instance, by implication of the middle ear and eventually the ossicles, or petrous portions of the temporal bones.

Scrofulosis of the eyes, the so-called *strumous ophthalmia*, usually begins with redness and swelling of the palpebral mucous membrane, and in the majority of cases is soon followed by involvement of the cornea, in the form of *phlyctenular keratitis*, with strong lacrimation, pain, and photophobia. The phlyctenulæ are very slow in healing, and show a great tendency to leave behind corneal opacities. Blepharoadenitis, madarosis and permanent thickening of the edges of the lids are quite common accompaniments.

Fig. 114.—Tuberculous axillary lymphadenitis.

The lymphatic glands are affected early or late—secondary to the inflammation of the skin and mucous membranes. Except their wide distribution, the glandular swellings present nothing characteristic in the beginning, but as the disease progresses they show a marked tendency to undergo caseation and suppuration. Furthermore, after evacuation of the pus which usually contains tubercle bacilli, they rarely cicatrize, but, on the contrary, continue as pus-discharging fistulæ or indolent ulcers.

The course of the disease depends greatly upon the vitality of the patient and the mode of treatment. It is always chronic. Children removed from the obnoxious surroundings frequently recover completely. In those not properly cared for, the tuberculous process is very prone to spread to the osseous system and to the internal organs. Spina ventosa, osteomyelitis and spondylitis form frequent sequelæ. (For details of these affections the reader is referred to the chapter on "Tuberculosis of the Bones," p. 761.) The internal organs, especially the liver, spleen and lungs, may be implicated singly or collectively, in which event the prognosis of course, is extremely bad.

Characteristic as the symptom complex of scrofulosis seems to be, errors of diagnosis are nevertheless very apt to be made. The perplexity is often great in the differentiation between scrofula and inherited syphilis, both of which diseases have many symptoms in common. In all such doubtful cases, it is wise, on the one hand to employ the tuberculin reaction, and examine the aural and nasal secretions as well as the pus from scrofulous abscesses for tubercle bacilli, and, on the other, to administer mercury and to look for the Spirochete pallida. One should not be too hasty in pronouncing a case as scrofulosis because of the so-called "torpid habitus" of the patient (pale, flabby, puffed face, thick nose, swollen and excoriated upper lip, redness and thickening of the lids), or the presence of adenoids or glandular swelling. These symptoms can and often do exist independently of tuberculosis, and, as already suggested, may be due to infection by other microorganisms.

Treatment.—Scrofula, like other forms of tuberculosis, demands early and energetic treatment. The patient should be removed from the obnoxious influences, should be well nourished and kept outdoors the greater part of the day. (See p. 449.) Under suitable conditions he should also be given the benefit of tuberculin therapy (*q. v.*). Internally we should administer, for several months in succession, moderately large doses of the syrup of the iodide of iron and syrup of the hypophosphites, as well as cod liver oil or similar alterative tonics. The local treatment, which is of very great importance, essentially consists of thorough bodily cleanliness (daily bath with sea salt, antiseptic dressings in open wounds, etc.) ; removal of diseased foci such as tonsils and adenoids, decayed teeth, caseated glands, etc., and evacuation of pus wherever found. Individual complications should be vigorously combated according to indications. (See bone tuberculosis, otitis, eczema, etc.) As the external lesions are probably carried from place to place by means of the fingers, open wounds (vaccination wounds!) should be thoroughly

protected and the patient's finger nails clipped and kept scrupulously clean to prevent scratching the diseased parts of the body and direct infection of its healthy portions.

R   Syr. Ferri Iodidi   ℥ iv   15.00
     Syr. Calcii et Sodii
         Hypophosphitum   q. s. ad f ℥ ii   60.00
     M.
     S.—One teaspoonful three times a day for a child
     three years old.

TUBERCULOSIS OF THE BONES AND JOINTS

Tubercular Osteomyelitis and Arthritis

The grouping together of tuberculous bone and joint diseases is intended to emphasize their correlation. The favorite seat of bone tuberculosis is usually in the epiphyses, the joint becoming involved

Fig. 115.—Tuberculosis of elbow joint in a boy eighteen months old. Note discharging sinus.

secondarily by extension of the inflammatory process to the synovial structures. Occasionally, the joint is affected primarily.

The immediate cause of the disease is the tubercle bacillus which invades the medullary tissue, the bone proper, or the articular structures, either from within—from a florid or latent tuberculous focus elsewhere—or from without, as a result of traumatism. An inherited predisposition and impaired nutrition from various causes favors the development of tuberculous disease.

Osseous, as well as articular, tuberculosis is essentially a chronic inflammatory process, free from the violent symptoms which are characteristic of acute, nontuberculous osteomyelitis. Extensive lesions may exist for weeks and months with apparently perfect health. Fever is usually absent in the beginning and only slight—in the evening—at a later stage of the disease. As the tuberculous process advances, progressive anemia and emaciation make their appearance, but are not pathognomonic of the affection. The local symptoms also are very vague at first. Hence the reason why local tuberculous disease is frequently overlooked until, as will presently be shown, deformity and loss of function have occurred, which vary greatly in extent and severity with the seat of the lesions and the mode of treatment.

Tuberculosis of the Vertebral Column

(Spondylitis. Pott's Disease)

This tuberculous process usually begins in or near the vertebral body, and if not arrested, gradually extends to the contiguous structures, including the spinal cord.

It is manifested by an ulcerative and often suppurative destruction of the bone, with metastatic (gravitation) abscesses in distant locations, e. g., retropharyngeal abscess, in cervical spondylitis; psoas abscess, in lower dorsal and lumbar disease. Furthermore, with softening and crumbling of the vertebral bodies, the spinal column, as it were, topples over, usually backward, producing a deformity known as kyphosis, gibbus or Pott's hump. The condition is gradually further aggravated by compensatory spinal deformities (especially lordosis) and a group of other distressing pressure symptoms, soon to be related, which if not arrested throw the unfortunate creature in an abyss of everlasting misery.

This process, fortunately, is very slow in development, affording ample time—from three to ten years—to arrest and mend its ravages and ample warnings to the patient to seek relief. We may frequently differentiate four stages in the progress of the affection: (1) The stage

of onset, where the symptoms are very vague and inconstant. The child shows a disinclination to play, refuses to walk, or tires easily when he does walk. He complains of pain in different parts of the body, following the distribution of the spinal nerves, the pain being often so severe, especially at night, that it wakes the child from his sleep with a sudden start— ''starting pain''; (2) the stage of fixation of the spinal column; (3) the stage of characteristic deformity; and (4) the stage of suppuration and pressure paralysis. The disease does not always progress to the last stages. In some instances, after two or three years' course, either through treatment or spontaneously, solidification of the
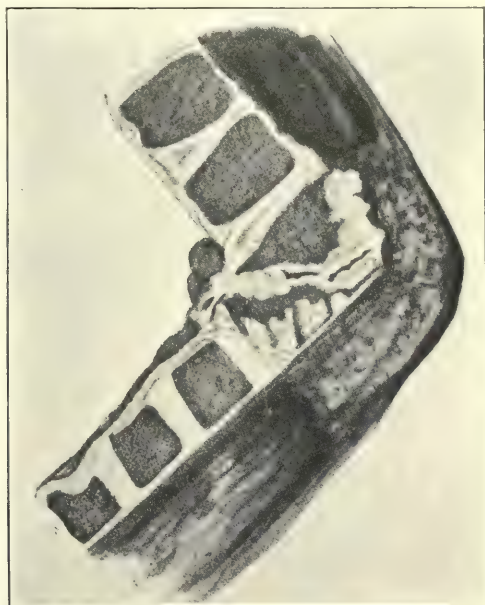
Fig. 116.—Pott's disease (Langerhans and Brooks, F. A. Davis Co.). Kyphosis of dorsal vertebræ, the result of caseous tuberculous periostitis and osteomyelitis. Destruction of three thoracic vertebræ. Two-thirds natural size.

diseased vertebræ—a relative cure—occurs. Relapses, however, are not infrequent. Pressure paralysis (see "Myelitis," p. 656) is especially common in disease of the lower cervical and upper dorsal regions.

The focal symptoms vary with the seat and extent of the lesion. In *cervical* spondylitis the patient, if old enough, complains of neuralgic pain in the head and upper portion of the neck. Very young children indicate the presence of pain by a suffering and an anxious expression of the face, by refusal of food and crying on handling. The head is stiff, tipped backward or laterally (torticollis-like), and, when the

child moves, he is often seen to support his head with the hands. At a later stage of the disease, there are often disturbances of deglutition and phonation, not rarely due to tubercular retropharyngeal abscess. If the uppermost cervical vertebræ are diseased, there is danger of anterior displacement of the head between the atlas and axis, more rarely between the occiput and atlas, and death from pressure upon the cord. The permanent deformity in cervical spondylitis usually consists of

Fig. 117.   Fig. 118.

Fig. 117.—Rigidity of neck associated with ''cervical ribs.'' For over two years patient was treated by eminent orthopedic surgeons for cervical spondylitis until a roentgenogram revealed the error in the diagnosis.

Fig. 118.—Same case as in Fig. 117 showing peculiar attitude of head which led to the erroneous diagnosis.

thickening and broadening of the neck, and sinking of the head upon the shoulders.

In *dorsal* spondylitis the distribution of the pain differs somewhat with the particular part of the spine involved. If the *upper* dorsal vertebræ are affected, the pain resembles that of intercostal neuralgia, and increases on coughing, sneezing, laughing, etc., while in spondylitis of

the *lower* dorsal vertebræ, the most frequent seat of the disease, the pain radiates to the lower extremities. In disease of this region, furthermore, the upper part of the body deviates to the side, one shoulder is elevated and the trunk bent to the opposite side—a state of scoliosis; at the same time the vertebral column is kept rigid, every movement carefully avoided, and, in walking, short rigid steps are taken, the patient timidly balancing the superincumbent weight of the body by firmly supporting the spine with the hands. If urged to pick up something from the floor, the child stoops by strongly flexing the knee-
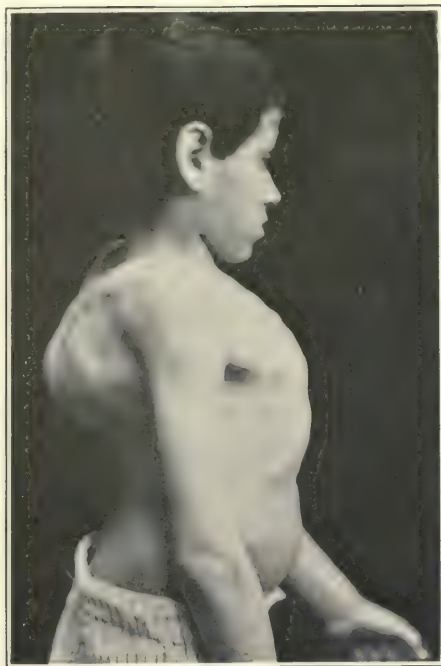
Fig. 119.—Advanced dorsal spondylitis with gibbus.

and hip-joints, while holding the vertebral column perfectly rigid, and raises himself by resting the hands upon the thighs, and then, with alternating supporting movements along the thighs and trunk, elevates the body and lastly extends the legs. If bending of the spinal column is attempted, motion occurs only in the healthy sections, the diseased portions remaining firmly fixed. The ultimate spinal deformity consists of kyphosis, kyphoscoliosis and lordosis.

In *lumbar* disease the patient complains of pain in sitting, and refers it also to the lowest portion of the abdomen and the legs. The physical

signs are essentially the same as in spondylitis of the lower dorsals, except that the deformity occurs at a later period and is not as pronounced. On the other hand, there is a greater tendency toward the formation of *psoas abscess*— a tumor deep in the iliac fossa or at the anterior surface of the thigh, lameness and flexion of one thigh.

Careful attention to the aforementioned physical signs rarely fails to disclose the presence of vertebral caries, even at an early stage of the disease. Cervical spondylitis may be mistaken for *torticollis* (sudden onset, pain and unilateral contracture more pronounced; early response to anodynes and antirheumatics, etc.); for *cervical rib* (revealed by x-ray); *nontuberculous retropharyngeal* abscess (immediate relief on puncture). Dorsal and lumbar spondylitis may be confounded with *rachitic curvature* (rounded in rickets; angular in spondylitis: rachitic kyphosis is reducible by placing the child upon the abdomen and overextending the thighs; absence of characteristic gait and mode of stooping). Right iliac psoas abscess often resembles *appendicitis* (onset sudden or recurrent, rigidity of the abdominal muscles, absence of spinal disease). Psoas abscess differs from *hip-joint disease*, by the hip-joint being fixed in the latter affection; and from *hernia*, by the latter being reducible in recumbent posture.

In view of the comparatively slow course of the disease in the majority of cases, the prognosis as to life is good, and as to permanent deformity fair, *provided* the treatment is begun early and persisted in. The prognosis is bad in cases presenting abscesses, fistulæ, and pressure paralysis. Even here surprisingly good results are often obtained under suitable treatment.

Treatment.—The treatment is principally orthopedic and surgical— fixation of the spine by a plaster of Paris or (in milder cases) celluloid jacket, rest in bed to unburden the spinal column, and evacuation of large collections of pus (*e. g.*, retropharyngeal or psoas abscesses). The F. M. Albee method of bone-grafting has proved eminently successful in a great many cases. Good hygiene, outdoor air, plenty of nutritious food, and iron, hypophosphites and cod liver oil will facilitate a cure.

Morbus Coxarius

(Hip-Joint Disease. Coxitis Tuberculosa. Articular Osteitis of the Hip)

The pathologic process of this tuberculous affection is usually described as consisting of three stages (1) The stage of ostitis, as a rule, involving the femoral head, less frequently the acetabulum; (2) the

stage of arthritis or suppuration, in which all the joint structures are implicated; and (3) the stage of disintegration and absorption of the head and sometimes the neck of the femur and the upper and back part of the acetabulum, with "wandering" of the head of the femur upward and backward upon the dorsum ilii.

Simultaneously with the onset of the first stage of the pathologic process, or sometimes at a later period, the child begins to limp and to

Fig. 120.—Tuberculous coxitis, advanced stage.

complain of pain in the knee- or hip-joint or both. As a rule, the limp at first is intermittent in character, more marked either in the morning or in the evening, but as the inflammation progresses, it becomes constant and quite pronounced, the leg at the same time being held very rigid. With the occurrence of articular exudation, the leg assumes a pathognomonic position of flexion, abduction and eversion, and the patient in order to bring the foot to the ground depresses the pelvis on the affected side, this giving rise to slight—apparent—lengthening of

the limb. With destruction of the joint and the articular bony structures, the hip-joint becomes further flexed, inverted and adducted. To overcome the uselessness of the limb in this position the patient elevates the pelvis on the affected side, and to counteract the—apparent—shortening, he steps on the ball of the foot. Later real shortening ensues, owing to the wandering of the femoral head upward and backward, and the firm contraction and atrophy of the muscles.

Fig. 121.—Early stage of hip-joint disease.

In consequence of the pelvic obliquity, in the upright posture, the patient assumes a position of compensatory scoliosis and lordosis. In the recumbent posture, with the limbs brought down parallel to each other, there is always compensatory lordosis of the lumbar region. This lordosis disappears on flexing the affected limb at the hip to an angle at which it is held flexed by the contracted muscles.

The intensity of the pain varies. It is usually worse after manipulation and fatigue, and at night. It may awaken the child from his

sound sleep with a cry (*"starting pain"*). The pain not rarely is referred to the knee, or to other parts supplied by the obturator nerve, *e. g.*, the inner side of the thigh. Hence the importance of always examining the hip-joint in such cases.

In addition to the pain, the limp and false position, we may find, at a late stage of the disease, involvement of the inguinal glands, with or without suppuration and perforation; enlargement—"white swelling" —of the hip; flattening of the gluteal region and effacement of one gluteal fold; multiple abscesses and fistulæ at various points of the hip or thigh, especially at the tensor fasciæ latæ, and irregular temperature, especially during the stage of suppuration.

Cases presenting the aforementioned typical symptoms are recognizable at a glance. Indeed, at this very late stage of the disease, it is almost immaterial whether a correct diagnosis is made or not, since a fatal issue from exhaustion, amyloid degeneration and general tubercu-

Fig. 122.—Hip-joint disease. Note compensatory lordosis on full extension of affected limb.

losis is all that can be expected, particularly in children with a tuberculous diathesis. The center of the physician's interest, therefore, should rest upon the diagnosis of incipient coxitis, which, if properly treated, offers good prospect of recovery. A history of slight trauma; occasional dragging of the leg or limping; pain in the hip- or knee-joint; disinclination to play and undue fatigue after slight exertion; restless sleep and "starting pain," all point to coxitis and demand very careful and repeated examinations of the hip-joint. The diagnosis is greatly facilitated and, in the majority of instances, rendered positive by the presence of pain on pressure against the trochanter, or against the acetabulum (by digital rectal examination), and by von Pirquet's tuberculin test. Advanced coxitis can readily be diagnosed by the aforementioned faulty attitude of the patient, in recumbency, standing, or walking. In doubtful cases, an x-ray examination (by an experienced

radiographer) is decisive. The latter procedure is especially useful in differentiating coxitis from: *Injury to the hip* (disability follows immediately after the accident; local signs of injury, *e. g.*, ecchymosis, etc.); *coxa vara* (x-ray shows downward inflexion of the neck of the femur; adduction and extension of the limb are usually possible); *congenital dislocation of the hip* (history of lameness from birth; absence of inflammatory signs or limitation of motion); *osteomyelitis* with separation of the epiphyses (very violent course); *rheumatism* (yields to the salicylates; no bone lesion); *spondylitis* of the lumbar region (distinct symptoms of spondylitis; hip-joint free); *hysteria* (absence of joint trouble, best proved under anesthesia, and by means of x-ray); *periosteal sarcoma* (Fig. 476) of the trochanter (the swelling rapidly increases in size; marked dilatation of the superficial veins).

Treatment.—The treatment consists of reduction of existing deformity, either gradually (by weight and pulley, while the patient is in bed) or forcibly (under anesthesia); disencumbrance of the hip-joint of the body weight, at first by rest in bed (bed extension apparatus) and later by means of an extension-walking apparatus (to enable the patient to enjoy fresh air) and, finally, fixation of the hip-joint by a plaster-of-Paris spica or a fixation apparatus. Fixation of the joint as well as extension should be continued for some time after apparent recovery. Constitutional treatment. Massage to prevent atrophy of the muscles and stiffness of the healthy joints.

Knee-Joint Disease

(Tuberculosis of the Knee-Joint, White Swelling)

The pathologic process of tuberculosis of the knee-joint resembles that of the hip. It may begin in the synovial membrane or in the articular ends of the osseous structures. The clinical symptoms are practically the same, whether the synovialis has been affected primarily or secondarily. They consist of fusiform swelling, local tenderness, atrophy of the thigh and calf muscles, flexion and slight outward rotation of the knee, and later abscess formation (extra- or intra-articular). During the suppurative stage, less frequently in the absence of suppuration, there are more or less constitutional symptoms, such as anorexia, anemia, emaciation and irregular fever. The latter is quite high in the presence of secondary infection.

The tuberculous process pursues a rather slow course. Not rarely it is interrupted by prolonged remissions. Exacerbations are often induced by local trauma or intercurrent acute diseases, sometimes after

an "apparent" cure has been established. The prognosis, as a whole, however, is favorable, if treatment is begun early and properly. The very rarely occurring spontaneous recovery should not be depended upon.

Treatment.—Within recent years the treatment of tuberculosis of the knee-joint, as well as that of the other smaller joints, has been en-

Fig. 123.—Tuberculosis of the knee in a thirteen month old infant who a few months later succumbed to tuberculous pyothorax.

tirely revolutionized. Instead of resorting to immobilization, resection and permanent fixation, Bier's method of passive hyperemia has become the treatment of choice, since it not only aids nature in the healing of the tuberculous process but tends also to restore the normal functions of the affected joint. The mode of procedure is very

simple. A soft rubber bandage about 2 inches in width is applied gently and evenly around the extremity (over a light flannel bandage), at some distance above the lesion, e. g., at the middle or upper third of the femur in tuberculosis of the knee-joint, and left in place for an hour or two, once or twice a day. If the bandage is properly applied, it gives rise to no pain, or interruption of the pulse. The extremity below the bandage soon swells slightly, and assumes a bluish-red color, but remains warm. The favorable results obtained from this mode of treatment of tuberculous joints are rather slow in coming (from three to nine months), but in uncomplicated cases well worth waiting for. Complications arising should be treated symptomatically. Thus cold abscesses call for free incisions and evacuation (may be enhanced by suction with Bier's cup) of the necrosed tissue; large exudations should be treated by aspiration and injection of iodoform emulsion or bismuth paste* and the general health should be improved by outdoor fresh air, nutritious food, tonics (iron and cod liver oil), massage and hydrotherapy. (For differential diagnosis, see "Arthritis," p. 418.) Tuberculin therapy (q. v.) is of undoubted value in the early stage of the affection.

Spina Ventosa

(TUBERCULOSIS OF THE METACARPALS AND PHALANGES, TUBERCULOUS DACTYLITIS)

This disease most frequently affects the first phalanx of the index finger, but may occasionally be found simultaneously in several phalanges or metacarpals of the same hand. The osseous tissue is gradually destroyed, and while this is going on, here and there new bone tissue is gradually formed under the periosteum. In consequence of the latter process, the finger becomes fusiform, as if the bone had been "blown up" (see Fig. 124). As the inflammatory process is very slow and painless, it, as a rule, takes several months before the characteristic appearance is developed. At a later stage of the disease, there is circumscribed redness, fluctuation, impairment of function of the tendons and spontaneous rupture of the suppurating focus with very tedious discharge of the contents.

Tuberculous dactylitis may be mistaken for a congenital or acquired syphilitic lesion. The history of syphilis, the presence of other syphilitic symptoms, the greater tendency of syphilitic dactylitis to be mul-

---

*Emil G. Beck's Method: Bismuth subnitrate 33 per cent, petrolatum (yellow) 67 per cent. The paste is injected slowly by means of a strong glass syringe with a conical pointed nozzle similar in shape to that of the ordinary urethral syringe, but much larger. Its prolonged use may give rise to bismuth poisoning!

tiple and symmetrical, and the ready response to antisyphilitic treatment usually suffice to clear up the diagnosis. A negative Wassermann reaction and a positive von Pirquet tuberculin test, and the coincidence of tuberculous lesions elsewhere, strongly point to tuberculosis.

Fig. 124.—Spina ventosa.

Early constitutional treatment including tuberculin (*q. v.*), and passive hyperemia (see p. 472) are very efficient curative measures. Conservative surgery (evacuation of pus and sequestra and injection of bismuth paste) is indicated in neglected cases. In these recovery is slow, usually with permanent deformity.

Nontuberculous Osteomyelitis

(OSTEITIS; PERIOSTITIS)

The term osteomyelitis refers chiefly to inflammation of the marrow of the bone, but includes also the morbidity of the bony matrix and periosteum, which at one period or another participates in the destructive processes.

Osteomyelitis is exceedingly common in children below the age of puberty—before completion of ossification of the epiphyses and diaphyses—since the anatomic peculiarities of the circulation in growing bones particularly favor its development on slight provocation. The affection is observed in two forms: nontuberculous and tuberculous (see p. 461). Nontuberculous osteomyelitis most frequently affects the long bones of the lower extremity (femur and tibia), less often the

other long bones, and exceptionally the short bones of the body. In most instances it is the result of infection of the medullary tissue by pus microbes, especially the staphylococcus and streptococcus, which enter the blood from suppurating wounds of the skin (pustular eruption!) or from pathologic foci in the respiratory or alimentary tract. As predisposing and contributory causes, we may mention the various contagious and infectious diseases, such as typhoid, scarlatina, measles, pneumonia, sepsis neonatorum, etc., all of which being instrumental in lowering the vitality and resistance of the patient.

Infection of the medullary tissue once established, the pathologic process is very acute and violent. If left alone, the inflammatory process rapidly goes on to suppuration, leading to loosening of the periosteum and bone necrosis and separation of the diaphysis from its epiphysis. If the patient survives and the inflammatory process subsides, there is a separation of the dead bone (sequestrum) from the living. Unless removed, the sequestrum may remain an everlasting source of irritation and suppuration.

The osteomyelitic process is usually ushered in by a chill, rapid rise of temperature and pulse and other symptoms which usually accompany acute suppurative affections. Before the appearance of the local symptoms, the disease is very apt to be mistaken for a pyemic or typhoidal condition; and in infants unable to indicate the presence of local pain, osteomyelitis may end fatally before a correct diagnosis has been arrived at. Hence, the importance of a careful examination of the bony system in all febrile affections with indefinite source.

In the newborn the onset may be insidious. There may be signs of omphalitis or the umbilicus may already be healed. For some obscure reason the infant may begin to cry, especially when handled. On careful examination it is found that the baby refuses to move the affected extremity (see Fig. 55).

The local symptoms of osteomyelitis are pain, tenderness, swelling, redness, synovitis, epiphyseolysis, and loss of function.

The pain is excruciating, boring or throbbing, worse at night, and increases in intensity as the exudation becomes more abundant. Young children are rarely capable of locating the exact seat of the pain, but usually refer to the entire affected limb. As a rule, the pain disappears suddenly with the escape of the inflammatory products from the interior to the exterior of the bone.

Tenderness on pressure can be detected early, and is most severe where the inflammation has approached nearest the surface of the bone.

When the disease is located deeply in the medulla, tenderness can be elicited by percussion.

Swelling and redness are not discernible until the inflammation has reached the periosteum. Thrombophlebitis and edema, however, are often early symptoms.

Synovitis is the rule where the disease affects the epiphysis as well as the end of the diaphysis. The intraarticular effusion is at first serous, the result of vascular disturbance, but as the suppurative process in the bone advances, the effusion becomes purulent by direct extension of the infection. The character of the effusion can readily be determined by exploratory puncture.

Epiphyseolysis, or separation of an epiphysis from the diaphysis, is a late symptom, or rather a complication. It may be recognized by soft crepitation between the separated parts, false point of mobility and displacement—signs of fracture.

Loss of function of the limb is invariably present, and as the disease advances there are marked contractures. The patient instinctively assumes such postures as will best relax the muscles and ligaments connected with the affected area, and thus prevent painful tension.

These symptoms, if closely kept in view, will generally avoid errors in the diagnosis. Typhoid fever can readily be excluded even before the development of local symptoms by the presence of marked leucocytosis in osteomyelitis. (For differential points between osteomyelitis and arthritides, see p. 418.) In cases of doubt a roentgenogram will almost invariably settle the diagnosis.

Treatment.—As previously indicated, the course of the disease varies with the degree of infection and the aggressiveness of the treatment. Early operative interference is usually followed by recovery in the great majority of cases. In some cases the infection is extremely violent and death occurs within the first thirty-six hours, before or notwithstanding that a diagnosis had been made and the appropriate therapeutic measures employed. The great danger in osteomyelitis is the tendency to venous and arterial thrombosis with secondary embolism and abscesses in different parts of the body, especially in the lungs, heart and kidneys.

With subsidence of the acute symptoms, the osteomyelitic process is not always at an end. Transition into *chronic* osteomyelitis is not uncommon. (For details, see a treatise on surgery.) Suppurating sinuses leading down to the infected sequestra may indefinitely persist, and, with occasional improvement, continue to undermine the vitality of the patient. Amyloid disease of various viscera (liver!) may form a sequel of prolonged suppuration.

Osteosarcoma

Next to the kidneys (see p. 582) the osseous structures, and more particularly the long bones, form the most frequent seat of sarcoma in children. The sarcomas may be of central or peripheral origin. The periosteal variety is usually more rapid in growth and more apt to in-

Fig. 125.—Osteosarcoma of the head and upper third of shaft of humerus in a boy ten years old. Note also early metastasis in the lungs.

volve the bone some distance from the joint. The etiology is still obscure. In the majority of instances we can elicit a history of traumatism at the seat of the tumor or its immediate vicinity.

In the early stages the affection may be mistaken for osseous tuberculosis, syphilis, or chronic periostitis. In tuberculosis the swelling

Fig. 126.—Enchondroma of upper third of humerus in a child eleven years old.

is more gradual in its development and most frequently attacks the joints. In syphilitic growths we usually find other signs of syphilis and a positive Wassermann reaction. Chronic or subacute periostitis with marked thickening of the periosteum and only a small amount of pus can readily be distinguished from osteosarcoma by a careful
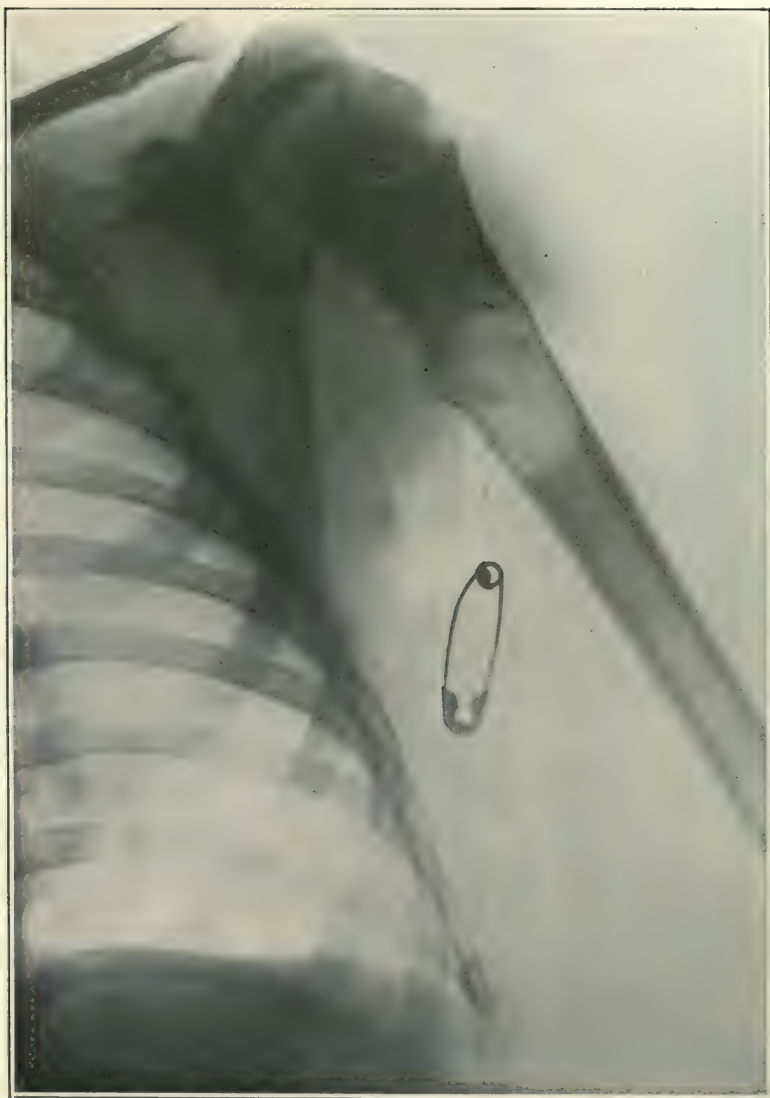
Fig. 127.—Bone cyst in shaft of humerus causing fracture in a child six years old.

Roentgen-ray examination, which should invariably be resorted to in cases of doubt. Of the benign neoplasms chondroma is most likely to simulate sarcoma, but chondroma is much slower in growth and unaccompanied by metastases—just the opposite of what is observed in sarcoma. In one case under our observation (Fig. 125) sarcomatous foci in the lungs were noted six weeks after the earliest signs of the tumor

in the humerus. Constitutional symptoms usually set in late in the course of the disease—hence the reason why the relatives of the little patients are loath to accept the physician's advice regarding early radical therapeutic measures, and hence also the extraordinarily high mortality.

Fig. 128.—Sarcoma of the left femur in a girl eight years old.

Treatment.—Where the diagnosis is made very early, *i. e.*, before there are any evidences of metastases, amputation of the affected limb, as high as possible above the seat of the lesion, may save the patient's life. The views on the results of radium treatment are too conflicting to take any chances on the postponement of the radical operation while awaiting the doubtful outcome of the radium treatment. All other methods of treatment thus far recommended are practically futile.

Scoliosis

(LATERAL CURVATURE OF THE SPINE)

In contrast to the aforementioned tuberculous deformity (spondylitis), scoliosis is not tuberculous in nature. As a rule, it is habitual, or static, the result of unequal (one-side) compression of the intervertebral cartilages, favored by atony of the muscles and ligaments and weakness of the bones. It is most frequently observed in school children, especially girls, and is generally ascribed to faulty posture while sitting at the school desk, etc., and to the habitual carrying of heavy books with one arm. I firmly believe that a great many cases of so-called habitual lateral spinal curvatures originate during early in-

fancy in connection with rachitis (*q. v.*), are generally overlooked while the deformity is slight, and are detected later, at a time when the deformity does and would gradually get worse, whether or not the child goes to school. Of course, this view does not preclude the fact that faulty posture and encumbrance of one-half of the body hasten to aggravate the curvature. Less frequent causes are obliquity of the pelvis (*e. g.*, shortening of one lower extremity from birth or postnatal disease); unilateral paralysis (*e. g.*, poliomyelitis, progressive muscular atrophy); unilateral immobility of the thorax (*e. g.*, protracted extensive pleuritic effusion or adhesions); and unilateral sinking of the

Fig. 129.—Lateral spinal curvature; second degree.

thorax from traumatism or operations (*e. g.*, multiple fractures of ribs, resection of ribs in pyothorax). Very rarely scoliosis is congenital in nature, when, as a rule, it is associated with other congenital malformations.

Scoliosis is manifested first by elevation of one shoulder, and later by prominence of one hip and scapula on the same side and gradually increasing convexity of the spinal column and side. With further progress of the deformity, the spinal column presents two curves, in the

shape of the letter S—the primary curve, which is usually in the dorsal region, and the secondary or compensatory curve, usually in the lumbar region. Bad cases are occasionally complicated also by lordosis, deformity of the thorax and displacement of the heart and lungs, but are otherwise free from constitutional symptoms. It may here be noted that marked lordosis has been found to be a cause of orthotic albuminuria (*q. v.*)

Fig. 130.—Lateral spinal curvature, S-shaped scoliosis.

Treatment.—Fortunately, nowadays, with the greater attention being paid to the general health of children, these dreadful deformities are very rarely encountered. Many cases come under the care of the physician in the first stage of the disease which ordinarily yields to massage, calisthenics, fresh air, ample nutrition, general medicinal tonics, and, above all, removal of etiologic factors. Severer forms of scoliosis are often corrected by a plaster-of-Paris or celluloid corset—worn continuously for several months, and followed by massage and exercise to

strengthen the weak muscles. Fixed scoliosis can at best only be impeded in its further progress, but the damage done is frequently irreparable. Hence, the importance of early and energetic treatment, and particularly of prophylactic measures, which are especially effective in habitual scoliosis. Here the school physician and teacher are offered many opportunities to merit the gratitude of the community.

Syphilis Hereditaria S. Congenita

(SYPHILIS EMBRYONALIS OR FETALIS, SYPHILIS NEONATORUM, SYPHILIS HEREDITARIA TARDA)

Congenital syphilis is due to a specific microorganism, the *Spirochete pallida,* which is transmitted to the embryo or fetus either through the syphilitic semen (ex patre), ovule (ex matre), or maternal blood (at any time during pregnancy).

The great majority of syphilitic embryos or fetuses are aborted. The few that survive may pass through the syphilitic process in utero (syphilis embryonalis s. fetalis), or may maintain a good state of health during intrauterine life, be born in apparently perfect health, and develop the syphilitic manifestations soon after birth *(syphilis neonatorum),* or not until several years after *(syphilis hereditaria tarda).*

Syphilis Embryonalis S. Fetalis

The few babies who survive the syphilitic onslaught during intrauterine life and are born at or near full term present a ghastly sight. They are shriveled and shrunken, emaciated and disfigured, with barely a spark of life in them. They are often asphyxiated and usually die soon after birth. Postmortem examination may reveal pronounced pathologic changes in the lungs (fatty degeneration of the pulmonary alveoli—''pneumonia alba''); in the liver (interstitial hepatitis); in the spleen and pancreas (induration and gummatous deposit); in the kidneys and suprarenal glands (perivascular infiltration and anemia necrosis); in the thymus gland (cystic degeneration and abscess formation); and in the osseous system (epiphyseal osteochondritis after multiple fractures). The skin affection consists chiefly of ''pemphigus syphiliticus,'' a bullous eruption on a dusky red, slightly elevated base, with a sanguinopurulent content. It is usually localized on the palms of the hands and soles of the feet. Owing to extreme tenderness of the body (syphilitic myositis?) the infant is very restless, and cries pitifully when handled.

Fig. 131.—Congenital syphilis, baby three weeks old. Note excoriation of upper lip from the "snuffles." The navel failed to heal and on several parts of the body the skin was exfoliating. Note also peculiar deformity of the feet.

Syphilis Neonatorum

As previously alluded to, the infant may at birth appear perfectly healthy. He may continue to thrive, especially if fed on breast milk. Before long, however—usually after from about one week to three months—the clinical aspect changes materially. The baby begins to breathe noisily, especially when it nurses, "sniffles," becomes hoarse, or loses its voice entirely. The nurse or the weather is blamed for the baby's "cold in the head," until examination reveals that the syphilitic coryza is associated with swelling of the nasal mucous membrane and occlusion of the anterior nares by a seromucous or serosanguinolent discharge and incrustation. Inspection of the mouth and throat often discloses grayish-white patches (plaques muqueuses) upon the mucous membrane of the mouth and pharynx, more rarely papillomatous vegetations, and occasionally edema glottidis, which latter may lead to fatal termination. Not rarely the inflammation of

the nasal mucous membrane extends to the nasal periosteum and peri-
chondrium, arresting the development of the nasal bones, and giving
rise to the peculiar sinking of the bridge of the nose which is generally
designated ''saddle nose.'' This is rather a late manifestation.

The syphilitic manifestations augment from day to day. The skin
assumes a peculiar light- or dark-yellow (copper) color, is dry and
hard to the touch, and soon becomes covered by an eruption which
is typical for its multiplicity and variability. Almost every kind
of skin disease is represented. Papules, vesicles, pustules, smooth
and scaly patches, tubercles, wheals, macules, hemorrhagic spots, sim-
ple redness, scabs, ulcers, etc., vie with one another in their supremacy,
and rhagades surround the different external orifices of the body

Fig. 132.—Syphilitic pemphigus, especially marked on the soles of the feet. Note
also condylomata at vagina and anus.

(angles of the eyelids and lips at the alae nasi, anus, labia vaginae,
etc.). The hairy portions of the body also participate in the syphi-
litic process. The hair of the scalp, eyebrows and eyelashes rapidly
fall out and are very slow in returning. The nails undergo certain
alterations, such as thickening, claw-like deformities, suppurative in-
flammation (onychitis) and exfoliation (paronychia), the process not
rarely extending also to the phalanges (syphilitic phalangitis, *q. v.*).
In the majority of cases we find a bullous eruption which is pathogno-
monic of grave syphilitic infection, *i. e., pemphigus syphiliticus.* It usu-
ally sets in within the first week after birth as flaccid, yellow or brown-
ish vesicles, surrounded by an areola of dry epidermis or excoriation.
The bullæ vary in size from a pinhead to a cherry, burst readily and
discharge a seropurulent or serosanguinolent content. They are dis-

tributed all over the body, but particularly over the palms of the hands and soles of the feet—herein differing from nonsyphilitic pemphigus which but rarely affects these parts. In consequence of the inflammatory state of the skin, the superficial lymphatic glands are more or less implicated, the swelling often persisting long after disappearance of the primary cause. Enlargement of the epitrochlear glands, just above the internal condyle of the humerus, is especially common and of diagnostic importance. Special mention deserve also the syphilitic condylomata, especially at the anus and female genitals. They usually begin as simple papules and are gradually transformed into luxuriant growths.

With the aforementioned clinical findings in view, it requires no sage to solve the problem of diagnosis. Now, if the physician bases his judgment upon the symptoms presented, does not allow himself to

Fig. 133.—Congenital syphilis in eight-week-old baby. Note multiform eruption, rhagades, and exfoliation.

be led astray by spurious histories (omnis syphiliticus mendax!), but goes right ahead and employs suitable antisyphilitic measures (see p. 494), the chances of rapid improvement and ultimate recovery are very good indeed. Otherwise, the syphilitic process often violently runs its destructive course, attacks one structure after another, one organ after the other, crippling the hapless infant for life, if it unfortunately survives.

The osseous system hardly ever escapes involvement. As in fetal syphilis (q. v.), the syphilitic bone affection consists principally of an osteochondritis and sometimes caries and necrosis. There is an overgrowth of the cartilage between the epiphyses and diaphyses of the long bones, often giving rise to painful circular swelling in the epiphyseal region and separation of the affected limb (spontaneous

fracture), with consecutive loss of power (Parrot's pseudoparalysis). This process is usually (but not invariably) unilateral, herein differing from rachitis in which the epiphysitis is almost always bilateral. The skull presents enlargements (Parrot's nodes) of the parietal eminences and a buffer-like bossing of the frontal bone which is generally designated "hot-cross-bun" tumor. Occasionally the frontal bone appears either unduly convex and prominent (frons Olympian) or keel-shaped, (Fig. 137) with a central ridge and lateral flattening. These syphilitic manifestations are often associated with craniotabes, delayed (or premature) closure of the fontanelles and great brittleness of the milk teeth.

The liver is often the seat of cellular infiltration (interstitial hepatitis) or variously sized gummata, rarely large enough to be visible

Fig. 134.—Congenital syphilis in a six-week-old baby. Note maculopapular eruption on baby and on mother's hand.

to the naked eye. The liver is enlarged, hard and uneven to touch, but palpable through the abdominal wall only in advanced cases. Marked syphilitic changes in the liver frequently give rise to icterus, acholic stools, and bile-colored urine. On the other hand, mild forms of the disease are usually entirely free from symptoms.

Next to the liver, the spleen is most prone to suffer in syphilis. It is enlarged and readily palpable through the abdominal wall. Splenomegaly being of so common occurrence in early childhood, it is difficult to determine how much of this phenomenon is due to the syphilitic process and how much to other causes, especially rachitis. The younger the infant (under six months), the greater the probability of the perisplenitis being syphilitic in nature, especially

if the splenomegaly is associated with other syphilitic symptoms, such as "Parrot's nodes," condylomata, and ozena.

Syphilis of the pancreas is not demonstrable during life, but it has

Fig. 135.—Syphilitic dactylitis of right index finger in a child two years old. (Note normal left hand.)

repeatedly been proved—by postmortem examinations—that the pancreas is affected in a way very similar to that of the spleen.

The intestines also are not rarely affected. Intestinal syphilis is

manifested chiefly by ring-shaped indurations of the muscles and mucous membrane, leading to gradual constriction of the intestinal lumen. The pathologic process resembles that of "Peyer's patches." Clinically, intestinal syphilis gives rise to protracted diarrhea, often with fatal termination.

Syphilitic changes (perivascular cellular infiltration; gummatous deposit) are occasionally met also in the kidneys and suprarenals (paroxysmal hemoglobinuria; nephritis), in the heart (symptoms of myocarditis); in the lungs (pneumonia with slow course; spirochetes in the sputum), in the thyroid gland (struma); in the thymus (cyst or abscess); in the testicles (often greatly enlarged; hydrocele; arrested development), and in the ovaries (demonstrable postmortem; sometimes by rectal, bimanual examination during life).
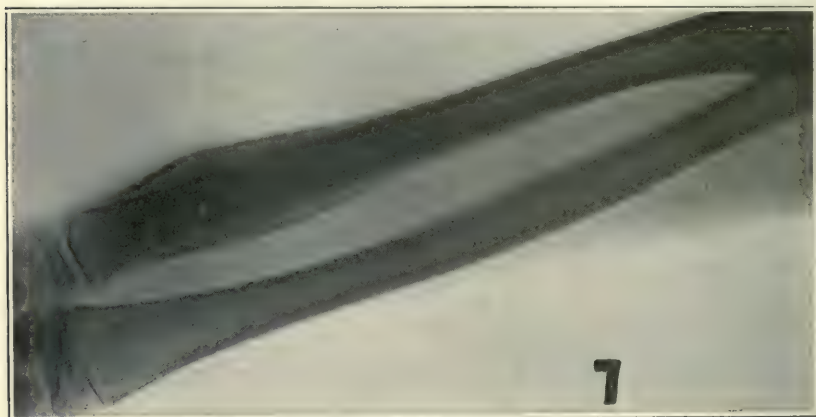
Fig. 136.—Periosteal syphilis of left ulna in a child ten years old.

Arteritis and periarteritis, gummatous deposits and sclerosis occasionally occur in the brain and spinal cord* as in the other organs of the body, and the concomitant symptoms vary with the seat of the lesions. Chronic meningitis and hydrocephalus with spina bifida are not rarely of syphilitic origin, and epilepsy, idiocy, local paralysis of the extremities and of the eye muscles, blindness, disseminated sclerosis and tabes dorsalis have been occasionally traced to congenital syphilis. Also cases of syphilitic encephalitis are on record. The resemblance between syphilis of the nerve system and tuberculosis should not be lost sight of.

As already suggested the diagnosis of syphilis is very easy when the aforementioned symptom complex is in full bloom. Cases, however, are not rarely encountered which are apt to test the skill of

*P. C. Jeans (Jour. Am. Med. Assn., Jan. 15, 1921) found involvement of the central nerve system in one-third of the cases examined.

even the best diagnostician. I am referring especially to those which either run a very latent course from the beginning, or do so after a few weeks' antisyphilitic treatment. Every bit of information as to the past, personal ("snuffles," eruption, etc.) and family history (miscarriages; persistent sore throat in the mother or father!) should be utilized to arrive at a correct conclusion. Old cracks and scars at the anus, mouth, nares, etc.; dark, mottled skin; old marks of healed ulcers in the mouth and throat; persistent ozena; intractible intertrigo, etc.; excessive brittleness of the milk teeth should all be carefully looked into, and where doubt still exists the patient be given the benefit of the doubt and actively treated for syphilis—the rapidity of

Fig. 137.—Syphilitic baby eleven months old. Note keel-shaped deformity and bossing of greatly distended head. Baby is also mentally deficient.

response to treatment at the same time serving as a differential point of diagnosis (therapeutic test).

Wherever possible laboratory tests should supplement ordinary clinical examination. Of these, Wassermann's serum diagnosis of syphilis and Noguchi's luetin intradermic test are deserving of special consideration. In cases of doubt, the parents should be tested as well.

With establishment of the diagnosis of syphilis, the remedies to be employed to eradicate the disease fortunately leave no room for speculation. The treatment, which will be fully outlined in the subsequent pages (see page 494), should be carried out energetically and systematically and continued until apparently every vestige of the disease has been completely removed.

Inadequate treatment not only greatly mars the prognosis of syphilis as to life and recurrences, but only too often is responsible for the development of the symptom complex which is generally described as "parasyphilis." This group of syphilitic manifestations (syphilitic cachexia) consists of extreme debility, marasmus (especially in the artificially fed); profound anemia (pseudoleukemia), obstinate gastrointestinal and bronchial catarrh, otitis (deafness), disposition to rachitis, cretinism and idiocy, and lowered power of resistance to divers acute infectious diseases. While the mortality of the carefully treated syphilitics is comparatively small, those who are carelessly managed often succumb to intercurrent diseases, even of the most trifling character, not rarely die suddenly without apparent cause, and if they survive, remain decrepit for life, and a source of horrible misery to future generations.

Syphilis Hereditaria Tarda s. Lata

Late hereditary syphilis attacks the offspring of syphilitic parents at any period between early childhood and adolescence. The children thus affected may or may not have shown manifestations of congenital syphilis during intrauterine life or soon after birth. The symptoms, however, are more pronounced in those who had been treated inadequately or not at all. Late hereditary syphilis essentially corresponds to the tertiary stage of acquired syphilis. Like the latter it shows a predilection for the eyes and osseous system; but no structure or organ of the body is free from its destructive effects.

As will be presently demonstrated, the lesions of late hereditary syphilis may be numerous and grave, but not always strictly pathognomonic of this disease. There is, however, one group of syphilitic manifestations, which, if present, invariably betrays the existence of a syphilitic taint.

This symptom complex is generally described as the "triad of syphilis" and consists of the following manifestations:

1. *The So-called Hutchinson Teeth.*—The characteristic teeth of syphilis are the two upper central incisors of the permanent set. The teeth are chalky, ill-developed, small, and irregularly placed. They taper from the free border to the base, hence the term "screw-driver teeth," and present a broad, semilunar notch in the center to the edge. They should not be confounded with the brittle and decayed milk teeth observed in infantile syphilis or rickets, and the irregularly implanted teeth associated with deformed palate or dental arches.

2. *Interstitial Keratitis.*—This almost invariably symmetrical affection begins with corneal haziness which rapidly increases until the entire cornea is in a condition of partial opacity resembling "ground-glass." It is associated with congestion of the ciliary region and slight inflammation of the conjunctiva, and in severe forms of the disease, with iritis, retinitis and choroiditis. In addition to the corneal gray-colored patches, abruptly margined, crescentic patches of salmon tint are often present on the corneal surface, this sign of vascularity not rarely spreading over the whole cornea and giving rise to a deep plum tint of purple redness. Excessive lacrimation and photophobia prevail from the start, in marked cases reducing the patient to a state of practical blindness. The disease runs a very slow course, from about three months to a year or longer, and, when it subsides, leaves behind more or less marked corneal opacity and visual impairment.

3. *Deafness.*—This condition is not accompanied by any inflammatory symptoms. It is caused by syphilitic involvement of the laby-
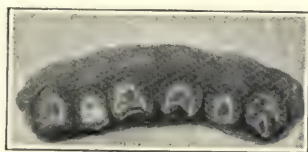
Fig. 138.—Syphilitic "Hutchinson teeth." Note semilunar notches in central incisors.

rinth (often deafness of both ears). The deafness very rarely clears up spontaneously and entirely. On the contrary, even under active treatment defective hearing is the rule. This peculiar form of deafness often precedes or follows the attack of keratitis and is gradual in development.

The *bone lesions* of late syphilis consist of an osteoperiostitis, or soft gummatous periostitis, especially of the tubular and cranial bones. The most frequent seat of the disease is the tibia, then follow the ulna and radius, the humerus, femur, clavicle, the bones of the skull, the phalanges and sternum. Syphilis of the shaft of the tibia usually gives rise to a characteristic "saber-shaped" deformity of the tibia, the so-called "tibia en lame de sabre." It differs from the rachitic deformity of the tibia by its crest being rounded (in rickets it is sharpened) and its internal and external surfaces convex (in rickets they are flat or concave).

The cranial bones are affected in a manner similar to that of syph-

ilis neonatorum. (See page 483.) Ulceration of the soft palate and throat, and perforation of the hard palate and nasal bones with secondary "saddle-shaped" deformity of the nose are of common occurrence.

Syphilis of the phalanges (syphilitic dactylitis) is characterized by a puffy, fusiform, or spindle-shaped swelling. It affects the fingers more often than the toes. The inflammation may begin either in the connective tissue and ligaments or in the periosteum and bone. If let alone the disease progresses rapidly and leads to protracted osteomyelitis with ankylosis, shortening and permanent deformity of the affected parts. Syphilitic dactylitis differs from the tuberculous

Fig. 139.—Gumma of the right parietal bone in an eight-year-old boy suffering from syphilis hereditaria tarda.

variety, which it greatly resembles, by its being less common, often symmetrical and accompanied by other syphilitic lesions. The tuberculin and Wassermann reactions are decisive in the diagnosis.

Occasionally *the joints* participate in the syphilitic process, but the affection is rarely of serious nature. It essentially consists of a recurrent synovitis with thickening and ankylosis, and may readily be mistaken for articular rheumatism. The absence of fever and redness and the history of syphilis usually clear up the diagnosis.

The *skin* sometimes presents subcutaneous gummata which, when neglected have a great tendency to break down and to form large phagedenic ulcers. They are most frequently met with on the face and upper part of the thighs or legs. They promptly yield to ener-

getic antisyphilitic treatment—a feature to be borne in mind in the differential diagnosis between syphilitic and tuberculous ulcers.

The *lymphatic* system and the viscera, especially the liver and spleen rarely fail to show late syphilitic manifestations. The latter are essentially identical with those described in connection with congenital syphilis neonatorum. (See p. 476.)

Finally, mention may be made of the tendency of late syphilis

Fig. 140.—Syphilitic osteoperiostitis of the tibiæ,—"Saber-shape-deformity,"—and of the nasal bones, with high degree of rachitis.

to arrest the development of the child's body and mind. Dwarfism and infantilism are not rarely traceable to this baleful cause. Indeed, appreciating the gravity, multiplicity and complications of the syphilitic lesions it is rather surprising that the aforementioned bodily and mental deteriorations are not more rampant.

Notwithstanding the apparent explicitness of the symptomatology, the diagnosis of late hereditary syphilis is by no means a simple

proposition. It is especially difficult in cases complicated by inter-current diseases, *e. g.*, tuberculosis or rickets.

The specific history; the simultaneous occurrence of lesions in various parts of the body; the tendency of the bone lesions to be symmetrical; the appearance of the manifestations very frequently in the midst of apparently perfect health; and, finally, the quick response to antisyphilitic treatment are more or less decisive in the diagnosis. Of course, all doubt is removed by positive microscopic or bacteriologic findings, especially serum diagnosis.

Acquired Syphilis

The newborn may acquire syphilis either intrapartum, by coming into contact with a chancre in the parturient canal, or while nursing at the breast of a woman (mother or wet-nurse) in the contagious state of syphilis. The disease may further be acquired by infants and older children practically in the same manner as by adults. It is well to remember that the newborn with secondary symptoms of syphilis may transmit the disease to healthy people through fondling, the use of articles coming in contact with syphilitic lesions, etc. I have in mind two older, previously healthy brothers, who have in this manner acquired syphilis from a syphilitic newborn.

The course of acquired syphilis in children is identical with that observed in adults, except that it is prone to be more rapid and violent.

The primary lesion (chancre) is usually found in the child's mouth (from kissing or sucking of infected nipples), or on the perineum (from washing of baby's buttocks with infected hands). Occasionally the primary sore is on the penis, as a result of infection during ritual circumcision.

Treatment.—The treatment of syphilis is alike in both forms of the disease—inherited (early and late) and acquired. It should be begun as soon as the diagnosis has been established. Temporizing is often fatal. Mercury in some form is the only remedy that is certain in its results, and should be administered *continuously* until every vestige of the disease has apparently disappeared, and then given *at intervals* of from two to six weeks for a total period of from two to three years. Calomel is the preparation *par excellence*. One-tenth to $\frac{1}{4}$ grain twice (to an infant) or thrice daily (to an older child) will usually suffice. Now and then we may also employ sodium iodide ($\frac{1}{2}$ grain for every year of the child's age) three times a day, or syrup of iodide of iron (5 drops for an infant under one year, 10 drops for two years, and 15 drops for over five). To hasten saturation of the

system with the mercury, we may, *in addition,* resort to mercury in-
unctions. From 10 to 30 grains of mercurial ointment may be rubbed
in once a day alternately into the axilla, groin, abdominal wall, calf
muscles, and loins. To prevent excessive salivation the oral cavity
should be washed twice daily with a 2 to 5 per cent solution of chlorate
of potash or tincture of myrrh. Syphilitic ulcers should be cauterized
with nitrate of silver solution (3 per cent to 10 per cent). Keratitis
calls for local use of atropine sufficient to keep the pupils widely
dilated, hot poultices (by means of moist hot cloths), occasional dust-
ing of calomel over the corneal ulcers, protection from bright light
(dark room or smoked glasses), and, of course, internal administration
of mercury and the iodides. The great majority of cases of osteitis
yield promptly to constitutional treatment, but where the necrosis
is pronounced the management must follow ordinary surgical lines.
Persistent condylomata will rapidly disappear after a few applications
of a 5 per cent salicylic-resorcin-collodion solution, or occasional
painting with caustics. Onychia and paronychia should be treated by
local bichloride baths (1:2000), once or twice daily, and dusting with
calomel 1 part, gum arabic 1 part, and stearate of zinc 10 parts. In-
durated lymph glands usually yield to potassium iodide ointment,
while suppurating glands require surgical interference.

The general health of the patient should not be lost sight of. Other
conditions being favorable, a syphilitic mother should nurse her
syphilitic child. This being impossible, the infant should be put on
properly modified cow's milk, or on the breast of a wet-nurse, who
has emerged from an attack of syphilis without serious consequences.
In older children also particular attention should be paid to good nu-
trition. The tendency of rickets complicating syphilis should be
borne in mind. Hydrotherapy, plenty of fresh, pure air, and general
tonics are essential to success.

Pediatrists are not particularly in favor of salvarsan in children, be-
cause it is difficult to administer it in young babies,* and is not at

---

*The Lowy Laboratory gives the following directions for the administration of neoarsphen-
amine in infants and older children:
Wrap the child in a blanket (as for intubation), lay it on the table on its side and place a
very low pillow under its head so as to have the head level with the line of the spine; then
sterilize the side of the neck. The external jugular vein will be found by drawing a straight
line from the outer portion of the lobe of the ear directly to middle of the clavicle. By placing
the little finger or a pencil a little above the clavicle directly in line with the clavicle, and exert-
ing gentle pressure, the jugular will become distended so as to become visible. Having properly
distended the vein, take a 20 gauge needle, insert it at the lower portion of the jugular vein
about one-half inch above the place where pressure is being exerted. With a firm push insert
the needle into the vein, care being taken, however, that the point of the needle is towards the
heart. When the blood begins to flow, without attempting to remove the needle, apply the
adapter supplied with Solution Arsphenamine-Lowy, and permit solution to flow in very slowly,
the dose being 20 c.c. per thirty pound body weight. It is sometimes advisable to measure out
the quantity desired into the barrel of the syringe and use the barrel of the syringe similar to
a gravity container, as in that fashion the amount used can be accurately gauged. Five to six
injections should be given at one week intervals. It may be necessary to give a series of these
injections.

all free from serious consequences, and, furthermore, because it shows no superiority over the mercury and iodide treatment. The dosage of salvarsan or neosalvarsan is 0.005 to 0.01 gram per kilo of body weight. It should be administered once or twice a week until the Wassermann reaction proves negative on several examinations. Syphilitic children should be kept under observation for several years after apparent recovery from the disease.

J. A. Fordyce* and I. Rosen combine mercury with neoarsphenamine treatment. The latter is administered intramuscularly, half of the solution into each buttock, in the following doses at weekly intervals: 0.075 gm. for infants from 3 to 8 weeks; 0.1 gm. from 2 to 6 months; 0.15 gm. from 6 to 12 months; 0.15 to 0.2 gm. from 1 to 2 years. These doses are employed in courses of from 6 to 8, followed by rest periods of from 4 to 6 weeks.

Frambesia

(YAWS)

Frambesia is caused by the *Treponema pertenue,* a slender spirillum resembling the spirocheta of syphilis. The mode of conveyance of the disease is still obscure, but probably occurs by direct contact. In endemic countries (Philippine Islands, Ceylon, Tropical Africa, Fiji, and Samoa) this affection is quite prevalent among young children. One attack seems to confer permanent immunity.

The incubation period lasts from two to five weeks, in the last few days manifesting itself by irregular temperature, muscular and articular pain, anorexia and lassitude. About a week later a papule makes its appearance which soon turns into a pustule, often perforated by a hair. On further growth this primary lesion assumes a raspberry-like appearance. The secondary eruption develops within from one to three months after the primary lesion, like the latter is preceded by general febrile symptoms, and like it also consists of cauliflower-like excrescences which may be distributed throughout various parts of the body but more especially over the face and neck and anal and genital regions. The lesions may recur at short or long intervals for years, especially if left untreated. Bone changes, especially in the feet and hands (dactylitis) are quite common; and as a result of extensive ulcers we occasionally meet with serious deformities of the hands and feet. Ulceration and necrosis of the frontal bone also is not infrequent. Cases presenting these symptoms may readily be mistaken for syphilis. Indeed, before the discovery of the Wassermann reaction some authors were inclined to look upon yaws as a type of syphilis. This view has proved erroneous, for it has frequently been shown that a patient with

*Jour. Am. Med. Assn., Nov. 20, 1920.

yaws may contract syphilis and *vice versa*. However, while frambesia is a disease *sui generis*, it has been found like syphilis to respond to intravenous injection of neosalvarsan. In the way of prophylaxis, it is important to avoid solution of continuity of the skin when handling patients suffering from the yaws, and to protect all abrasions by means of collodion, adhesive plasters and antiseptics.

Leprosy

Leprosy is an infectious disease pursuing a chronic course due to an acid-fast bacillus, the *Bacillus leprœ* (discovered by Hansen of Bergen in 1871) which resembles the tubercle bacillus. It is an uncommon disease, especially in sanitary countries. In the following table Dr. Heiser gives an approximate estimate of the proportion of lepers to the population of different countries.

Japan 1 in 1000.
Philippine Islands 1 in 1400.
India 1 in 2000.
United States 1 in 100000.
New Zealand 1 in 200000.
Australia 1 in 200000.

The exact mode of transmission of the disease is still unknown. It is not congenital in character. Fifty children born of leprous parents at the Culion Leper Colony showed no traces of leprosy at birth. Some of them, however, contracted the disease later, by remaining in close contact with their mothers. Kitasato maintains that over 7 per cent of the children of lepers sooner or later acquire the disease. The incubation period is of variable duration, in some instances several years. Once the disease is established it is found that the bacilli entering the human body have multiplied enormously and become enclosed in plasma cells (lepra cells).

We generally distinguish three varieties of the disease, as follows:

1. **Anesthetic Leprosy.**—In this form the anatomic changes (infiltration) occur principally in the nerve system. It ordinarily begins with shooting pain, particularly in the ulnar and peroneal nerves, flushing, erythema, of the face, glossy skin and muscular twitching. It is soon followed by anesthesia of large surfaces of the body. With further progress of the disease and consecutive destruction of the nerves, trophic changes, especially of the extremities soon supervene, accompanied by ulceration of the affected structures which fail to heal and gradually undergo total destruction (amputation of terminal extremities). This process is often associatetd with marked contractures, *e. g.*, of the thumb and fingers (*main en griff*).

2. **Tubercular, Nodular or Hypertrophic Leprosy.**—This variety is usually ushered in by a macular eruption and febrile disturbance. At first the ears, nose and face are infiltrated. Gradually the eruption assumes a nodular, tubercular consistency, resembling a crop of red potatoes, and spreads all over the body. In late stages the contour of the

Fig. 141.—Case of leprosy in a child showing infiltration especially in ears, lips and hands. Leprous nodules in the left arm. Example of tubercular, nodular, hypertrophic leprosy. (After J. C. DaCosta, Jr., Handbook Medical Treatment, F. A. Davis Co.)

face resembles that of a lion. Infiltrations occur also in the larynx, lungs and eyes, but perforating ulcers are not as common as in the anesthetic variety.

3. **Mixed Leprosy,** is characterized by the intermingling of the principal symptoms of the two other varieties of the disease, and is the most common type met with in leper asylums.

The diagnosis presents no difficulties in advanced cases, but in the early stages leprosy may be mistaken for lupus or syphilis, and in doubtful cases it may become necessary to search for the acid-fast lepra bacilli in the scrapings of the affected tissues, in order to arrive at a prompt decision.

Treatment.—Isolation and segregation of the patient in a leper hospital until at least two years after total disappearance of the clinical manifestations as well as the microscopic findings. Even then lepers should be kept under surveillance. Persons with open wounds should not come in close contact with lepers. The active treatment consists of hypodermic injections of the following preparation (Unna's modification):

℞ Chaulmoogra oil (obtained from the seeds of gynocardia odorata)*
    Camphorated oil   āā 30.0
    Resorcin   2.0
Mix and dissolve with aid of heat and filter.
Sig.—Five to fifteen drops hypodermically once a week. Quicker results
    are obtained when the injections are made in the infiltrated areas.

In cases where severe reactions follow (*e. g.*, fever, cardiac distress), the dose may be reduced in quantity but given more frequently. Prolonged hot bicarbonate of soda baths act beneficially. Ulcerations and other symptoms are treated according to indications.

Pestis Bubonica

(BUBONIC PLAGUE, BLACK DEATH)

The history of this dreadful scourge is traceable to the old Bible. In recent years it raged in China and India and sporadic cases were observed in port cities of Italy, Scotland and England as well as those of South America and the United States (New Orleans, Pensacola, Galveston, Seattle and San Francisco). Most recently several cases of the plague have been reported from Mexico and Texas.† The disease is spread mainly through infected rats, by bites of rat-fleas. It is caused by the *Bacillus Pestis,* which was discovered by Yersin and Kitasato in 1894 and is regularly found in the circulating blood of plague patients as well as in the sputum (pneumonic plague), enlarged glands, spleen and other organs of the body.

---

*Walker and Sweeney (Jour. Infect. Dis., 26, 1920) have recently demonstrated that this oil contains bactericidal substances that are about one hundred times more potent than phenol, and that its action is specific for the acid-fast group of bacteria.

†Analysis of 26 cases of Beaumont and Galveston, by M. D. Levy (Texas State Journal, October, 1920).

The incubation period ordinarily lasts two or three days, but occasionally may be of much longer duration. The attack is ushered in with chills, high temperature, mental depression, delirium and often convulsions. These symptoms are soon augmented by the appearance of painful swelling of the inguinal (*bubo*) and axillary glands, petechiæ (hence the name of "black death") and occasionally internal hemorrhages, and in fatal cases muttering delirium and coma. *Septicemic* plague with early prostration, vomiting and dysentery and low temperature, ends fatally even before the appearance of the bubo, while mild cases, *pestis minor*, may not be ill enough to go to bed and can only be diagnosed by the finding of the bacilli in the glands or blood. In about ten per cent of cases the plague appears in the form of pneumonia (*pneumonic* plague). The symptomatology of this type of the disease greatly resembles the so-called epidemic influenza pneumonia, characterized by extreme dyspnea, profuse bloody expectoration, cyanosis and heart failure, and is almost invariably fatal. It has often been observed that patients surviving six days show a tendency to recover, although convalescence is slow and not rarely marred by complications. The glands usually suppurate and either break spontaneously or have to be incised. The mortality as a whole ranges between 60 and 90 per cent. Postmortem examination usually reveals involvement of the spleen, lungs, liver, heart and kidneys, all more or less studded with small, confluent hemorrhages.

Treatment.—Prophylaxis is most essential. Rats and their breeding places should be promptly destroyed. All patients should be strictly isolated, and persons, as well as animals, coming in contact with them quarantined. Ships carrying suspicious cases should be detained. Infected buildings should be fumigated and if possible destroyed.

Prophylactic vaccines of Haffkine and others administered hypodermically in the arm twice at an interval of ten days affords sure immunity against the plague for about three months. It has recently been shown also that Haffkine's vaccine in combination with Tersin's serum (live bacilli injected into a horse) is potent to reduce the mortality to less than 20 per cent. Similar results are claimed for Lustig's serum with antitoxic properties (obtained by immunizing horses to the endotoxin of the bacillis pestis). The serums are given intravenously as well as in the affected glands.

Aside from the specific treatment special attention is devoted to the heart, nutrition and general comfort of the patient.

Physicians and attendants of plague patients, especially of the pulmonary type, should wear face masks of canton flannel with eyes of celluloid.

CHAPTER VIII

DISTURBANCES OF METABOLISM

Marasmus, Athrepsia, Infantile Atrophy

(PEDATROPHY)

The nature of this appalling infantile wasting is still shrouded in mystery. It is apparently only a functional disorder, a form of intestinal autointoxication, arising from nonassimilation of the food consumed, since the organic lesions (atrophic patches in some portions of the intestinal tract and indefinite degenerative changes in the lungs, liver and kidneys found postmortem) are not uniform, and rapidly disappear when the atrophic infant is put on a suitable diet, which may vary from an ideal breast milk to some proprietary artificial food (!). In this group, of course, are not included cases of marasmus accompanying tuberculosis, syphilis and the like.

Whatever the pathology and cause, the symptomatology is very pathognomonic. The apparently normally born infant, after thriving fairly well on the milk mixture it has been receiving, begins to show signs of ill health and arrest in weight. The food disagrees, it is occasionally vomited or regurgitated. The stools are either constipated, dry and soapy, or green and frequent, scanty in quantity, and contain undigested particles of food. The child suffers from colic, especially soon after feeding, is very restless, cries and whines pitifully, sleeps poorly, and do what you will, emaciation sets in and continues at a rapid pace. Before long the fontanelles, the eyes and cheeks are sunken (except for the small cushions of fat, "sucking pads," over the buccinator muscles); the nose and chin pointed; the abdomen is at first prominent but later retracted, the skin wrinkled, often hanging in folds, and, adding to this, the earthy pallor and senile expression of the face, the poor creature is a sight dreadful to behold. Though dried up to mere skin and bone, with respiration shallow and pulse bad, the infant keeps on fighting for life for weeks and months, not rarely successfully. On the other hand, sudden death may occur when least expected.

Unless wrecked by intercurrent diseases, those showing tenacity to life, and coming under observation not entirely in a hopeless state, stand some chance to regain their vitality and to recover com-

pletely. The prognosis depends also upon the duration of the marasmus, the age of the patient—it is more favorable in infants over four or five months than in younger ones—and the care he can receive from those in attendance. The concurrence of complications or sequelæ, such as atelectasis, edema, pneumonia, colicystitis, pyelonephritis, osteitis, general furunculosis and the like, greatly mar the chances of recovery.

Treatment.—As athrepsia almost invariably occurs in artificially fed infants, the line of treatment which at once suggests itself is to supplant the artificial food by human milk. Indeed, through such a change miraculous improvement in the infant's condition may often

Fig. 142.—Marasmus in a child ten months old. Note ''senile face.''

be observed within a very few days, requiring no further treatment to complete prompt and uneventful recovery. Wet nursing, therefore, should be the treatment of choice, even if it be only for a month or two, after which period cow's milk feeding may frequently be resumed with success. Athrepsia in breast-fed babies is usually due to an excess of fat or some other constituent in the milk. In such cases the difficulty may often be surmounted by allowing the baby to nurse only from five to ten minutes at a time, and giving it 1 or 2 ounces of plain or cereal water, or diluted lime water, before and after each feeding, or a light skimmed milk mixture after nursing. On the other

hand in some cases there is a deficiency in the food elements and the persistent inanition is responsible for the athrepsia. When the services of a wet-nurse are not obtainable (for financial or other reasons), an attempt should be made to feed the baby on condensed or dry milk in low dilution with plain or barley water. In a number of cases fat-free milk (1:1 or 2:1), in small quantities to begin with, proves useful, and to bridge over the critical period protein milk (1:1) may be tried, occasionally with splendid results. Finally, malt soup for reasons rather difficult to explain (unless it be assumed that the marasmus is the result of acid intoxication which is arrested by the carbonate of potash of the malt soup) will often, within a short time, convert a baby reduced to skin and bone into one of perfect beauty.

Lavage and colon irrigation are useful in all cases. The latter should be employed daily; the former every alternate day, or more often, if the return water contains large quantities of mucus, and the vomiting persists. In the latter event it is often of advantage to add a little boric acid or bicarbonate of soda to the sterile water used for stomach washing. Of medicinal agents, in addition to an occasional dose of calomel, pancreatin is the only remedy to place some reliance upon. One or two grains each of pancreatin and bicarbonate of soda may be administered after feeding.

The mouth of the infant should be kept scrupulously clean, and the buttocks dry and clean, to prevent stomatitis and intertrigo, both of which form frequent complications. The child should not be left too long in a recumbent posture, lest decubitus or passive pulmonary congestion supervene. For details of treatment of atelectasis, edema, and other complications the reader is referred to the respective chapters on the subjects.

Outdoor life and plenty of fresh air while the patient is indoors are essential to successful management of the cases in question. Whenever possible the child should summer in the country. Above all, however, breast milk is the specific for marasmus, in the way of prophylaxis as well as cure. (See "Tuberculosis" and "Syphilis".)

Rachitis

(Rickets, The English Disease)

Rickets is one of the most common affections of early childhood. It prevails to a greater or less extent in almost all parts of the world, but shows a predilection for poorly born, poorly nourished (also among the well to do) and poorly housed children of temperate zones. The immediate cause of rickets is the absence or deficiency of im-

portant elements* in the food or failure of the organism to assimilate the same in sufficient quantity. J. A. Schabad who has made a very exhaustive study of the subject (Arch. f. Kinderheilk., Vol. 54, Nos. 1 to 3), is of the opinion that the pivotal point in the morbid mechanism of this disease is the metabolism of phosphorus and not of calcium. In the evolution of rachitis there is an increased elimination of both of these substances. The increased excretion of phosphorus is greater than can be accounted for by the amount of this substance and the equivalent amount of calcium contained in the bones, so that it is probable that the

Fig. 143.—Rachitic "frons quadrata" in an infant thirteen months old.

nervous tissues share in the pathologic process. The increased phosphorus elimination is in the feces, while that in the urine is really less than the normal (hypophosphaturia). The ratio between the phosphorus content of the urine and that of the feces is changed. The normal ratio in nursing infants, 80:20, becomes in rickets 65:35, while in artificially fed infants and in older children the normal ratio of 60:40 is practically reversed to 40:60. In the convalescent period this disturbed ratio of phosphorus elimination is gradually restored to normal, while the total excretion of phosphorus reaches the subnormal. At the same time there is a relatively great increase in the urinary phosphorus, the

*Vitamines. (See p. 114.)

ratio becoming 75:25. There is a close relationship between the calcium and phosphorus content of the feces; increase in the intestinal elimination of calcium goes hand in hand with a phosphorus retention and *vice versa*. As its direct and most conspicuous result, we have a great diminution of the inorganic elements of the bones (barely 35 per cent, whereas in normal bone they amount to 65 per cent), exaggerated production of epiphyseal cartilage, excessive cell proliferation beneath the periosteum, and incomplete ossification of the new osseous

Fig. 144.—Rachitic beading of the ribs, "pot-belly," and bow-legs.

tissue. The same process takes place in the centers of ossification of the flat bones. This is especially true of the cranial bones, giving rise to areas of thickening (bosses) and relative thinning (craniotabes). As the disease advances, chronic inflammatory changes occur also in the different soft structures (muscles, arteries, etc.) and organs (spleen, liver, nervous system, etc.) of the body, leading to a complex pathologic entity *sui generis*—entirely distinct from any other diseased process.

This pathogenic process is very insidious in its onset and its course; hence in the beginning rickets is very apt to be overlooked, especially if following upon some other illness.

As a rule, the initial symptoms are very vague, and consist of recurrent indigestion, restlessness and debility: a nonpathognomonic group of symptoms, rarely arousing the anxiety of those in charge of the patient so as to seek medical advice. When seen by the physician, therefore, the disease is usually in full bloom.

The skull is relatively large, the forehead broad and prominent in profile (frons quadrata). The parietal eminences project strongly, and the fontanelles, especially the anterior one, and the sutures fail to close in due time. The occiput is thinly covered with hair or

Fig. 145.—High degree of rachitic spinal curvature.

entirely bald, and here and there yields to pressure with the finger (craniotabes). These soft spots are usually quite pronounced when the rachitis sets in during very early infancy.

The local baldness is the result of undue pressure and friction of the occiput against the pillow, and the effect of profuse perspiration which is most marked at the posterior portion of the head. The sweating and rubbing of the head, both very early symptoms of rickets, in a way are correlated, and probably due to cranial hyperemia. The rachitic process is also accompanied by more or less severe local pain in the bones, and the little patients will often cry when lifted

and are even annoyed by the pressure of ordinary bed covering (hence kicking off of the blankets).

The lower jaw, instead of being rounded, becomes flattened, and its alveolar edge is turned inward. The upper jaw is also more or less deformed, and the teeth, which are late and irregular in coming, are asymmetrically set, conforming with the altered shape of the jaws. Owing to the deficiency in enamel the teeth soon turn yellow, brownish or black, are streaked and brittle and subject to rapid decay.

The rachitic thorax is very typical in appearance. The clavicles are more sharply curved than in the normal, and occasionally infracted; the costochondral junctions are thickened, bead-like in shape (most marked from the fourth to the eighth rib), assuming in their sloping course from above downward a rosary-like appearance (rachitic rosary); the sides of the thorax are flattened and the sternum projects,

Fig. 146.—Rachitic bow-legs, ''jug''-shaped abdomen, and separation of epiphyses —''double-jointed.''

as in birds, hence the so-called ''pigeon'' or ''chicken'' breast (pectus carinatum), and, finally, the lower lateral diameter is widened.

The vertebral column, although rarely affected in mild forms of rachitis, invariably suffers in severe and protracted cases. The deformities most frequently met with are kyphosis and scoliosis. The kyphosis or backward curvature usually extends from the middorsal to the sacral region. It differs from tuberculous kyphosis by being rounded, and in the early stages reducible when the child is placed upon the abdomen and the thighs are overextended. (See ''Spondy-

litis,'' p. 462.)   Rachitic lateral curvature or scoliosis is produced by the relatively heavy weight of the head upon the yielding (muscular and ligamentous insufficiency) vertebral column.   The condition is further aggravated by allowing the patient to sit up or walk at too early an age and for too long periods, and by the habitual unequal distribution of the encumbrance.   As regards the latter, it will be noted that right-handed persons usually carry their children on the left arm, so as to have the right hand free, and in consequence, the right pelvis of the child is lifted upward, the right shoulder tilted downward and the middle spine shoved laterally—lateral scoliosis with the spinal convexity to the left.   While rachitic scoliosis is most frequently observed in early childhood, rickets undoubtedly forms also the principal cause of the so-called postural scoliosis of school children, the curvature being merely an exaggeration of the former condition.   Rachitic scoliosis (Fig. 145) is to be differentiated from congenital scoliosis (very rare; as a rule associated with other congenital deformities); cicatricial scoliosis (following operation for purulent pleurisy); paralytic scoliosis, in association with poliomyelitis, etc.   (Fig. 189); spondylitic scoliosis, usually kyphoscoliosis (see ''Spondylitis,'' p. 462); and static scoliosis (in congenital or acquired shortening of one lower extremity).   Although, as previously alluded to, rachitic scoliosis is reducible in its early stage, if let alone for a long period, the deformity is apt to remain permanent, notwithstanding the disappearance of the other symptoms of rachitis.

The extremities very rarely escape involvement.   In the upper extremities we usually find marked enlargement of the epiphyses at the wrists, and less frequently at the elbow.   In creeping infants the radius and ulna are often curved and sometimes infracted, and in severe cases the hand is separated as it were by a furrow—''double jointed.''   Occasionally there is also an enlargement of the ends of the metacarpal bones or the phalanges.

By far more marked are the deformities of the lower extremities. The soft tibia and fibula are ill prepared to balance the weight of the body.   The flimsy fundament thus tumbles under its encumbrance. The hapless patient learns to walk late and with difficulty, or, as it were, ''forgets'' or unlearns how to walk, or refuses even to stand because of pain in the legs.   If he continues to walk, the tibiæ and fibulæ bend either outward (bowlegs—genu varum; O-shaped) inward (knock-knees—genu valgum; X-shaped) forward (saber-blade shaped) or, in severe cases, simultaneously in different directions.   Similarly to what occurs in the upper extremities, there is also an enlargement of the epiphyseal ends of the bones, and occasionally infraction of the diaphyses.   Children sitting crossed-legged may present also more or less pro-

nounced curvatures of the femur and pelvis. Rachitic flatfoot is rare.

The course of these deformities varies. In the majority of mild and moderately severe cases spontaneous recovery occurs with improvement of the general condition. On the other hand, in extreme cases, where, as a rule, growth is greatly retarded, the curvatures persist and require forcible corrective measures, or surgical interference.

The muscles generally participate in the rachitic process. They are thin and flabby and partly responsible for the difficulty in sitting and walking ("pseudoparalysis"); abdominal distention ("potbelly"); and

Fig. 147.—Rachitic knock-knee in girl six years old.

for the constipation and prolapsus recti. The muscular insufficiency may be associated with overfatness, and mask the local rachitic manifestation.

The ligaments are more or less lax allowing undue mobility at the larger joints, and giving rise to the abnormality known as "double joints."

Coincidently with and in a measure because of the gross alterations in the body framework, manifold changes occur also in the functions and structures of other organs of the body.

The respiratory system suffers early. The contracted chest compresses its contents and disturbs the equilibrium of the thoracic and abdominal organs. The area of breathing space is reduced, hence, respiration is more or less interfered with, and the tendency to respiratory disease greatly increased. The latter is favored also by the

timidity of the parents to expose their delicate babies to outdoor air, keeping them huddled up in poorly ventilated rooms and thus reducing their power of resistance to infection. In consequence of this, slight catarrhal affections of the nasopharynx or larynx, instead of, as in the normal, yielding promptly to suitable treatment, they persist indefinitely and lead to capillary bronchitis or bronchopneumonia, not rarely with fatal issue or greatly protracted convalescence with a predisposition to tuberculous infection. As an immediate result we have also profound secondary anemia—reduction of hemoglobin and red blood cells and moderate leucocytosis. The child is pale, sometimes waxy in color; its digestion is poor; diarrhea alternates with constipation (often the feces are hard and the rectum is unable to expel the lump until aided by mechanical means), the latter, however, preponderating. The liver and spleen are more or less enlarged and help to distend the abdomen, sometimes to such an extent as to greatly resemble tuberculous peritonitis (see p. 155). Rachitic children are very irritable, sleep restlessly, and show a great disposition toward different spasmodic conditions. Spasmus glottidis, spasmus nutans, eclampsia and tetany are frequent complications of severe and protracted cases of rickets, especially in very young infants.

Cases of rickets presenting the local and general symptoms here depicted usually offer no diagnostic difficulties. Less typical cases, however, may be confounded with cretinism, achondroplasia, congenital syphilis, incipient hydrocephalus, and osteogenesis imperfecta—a group of diseases which not only have several symptoms in common and are to a certain extent etiologically correlated, but may also be associated with rickets.

In *cretinism* there is marked mental deficiency; the tongue thick and protruding from the mouth; as the child grows older there is very pronounced disparity between its age and body length.

Achondroplasia is characterized by a striking disproportion between the length of the trunk and extremities; the curvature of the shafts of the bones is due to embryonic defective development and not, as in rickets, to softness of the bones; the fingers do not lie parallel as in the normal, but are spread out like ribs of an open fan.

The epiphyseal thickening at the ribs and the long bones of *syphilis hereditaria,* as a rule is observed soon after birth in association with other symptoms of syphilis which yield promptly to specific treatment.

Incipient hydrocephalus has several symptoms in common with rickets (separation of the fontanelles, softening of the cranial bones, irritability of the nerve system). In hydrocephalus, however, the cranial distention is rapidly progressive in character, leaving the long bones of the body, which suffer most in rickets, almost unmolested.

Osteogenesis imperfecta differs from rickets in that in the former the bones are so soft that they can be cut and bent, splintered and fractured in several places.

The importance of an early diagnosis cannot be too strongly emphasized, since upon it depends the prognosis and the success of the treatment. While it is generally admitted that rachitis *per se* is not dangerous to life, and that in a number of cases spontaneous recovery is possible, the indifference of the laity as well as the physician regarding early and persistent treatment is strongly to be deprecated. Spontaneous recovery is rarely complete. On the contrary, without suitable treatment, the majority of children are left stunted in growth, distorted in shape and features, and depressed in spirit—in short, poorly qualified to struggle for an existence and to compete with the fellowmen favored by good fortune with sound mind and body.

Treatment.—Rickets is preventable by abundance of sunlight and fresh air and by a mixed, nutritious diet. In the absence of contraindications, children over three months of age should receive in addition to milk, small quantities of carbohydrates and orange juice; those over six months, also thin meat and vegetable soups; those over nine months, half of or a whole soft-boiled egg, some beef juice, and a little toasted bread with sweet butter; and those over a year, one egg daily, some thick fresh vegetable soup, with finely scraped beef or chicken, oatmeal gruel, light cocoa, etc., and occasionally a small quantity of finely scraped fresh beef spread on bread or mixed with baked potato. Season permitting, raw milk should be given in preference to boiled, sterilized or pasteurized.

Rachitic deformities may be prevented by avoiding superencumbrance of the spine and extremities. Infants with incipient rickets should, as much as possible, be kept off their feet, and advantageously held in the recumbent posture, allowing them to remain in the upright position only for short periods at a time.

The suggestions just made apply as well to the management of further advanced cases of rickets. Here, too, sunshine and nitrogenous diet in abundance and removal of the superincumbent weight of the body are the remedies *par excellence*. To these we should add hydrotherapy (sea salt baths), massage and passive motion, and corrective light braces where the deformities persist. Operative corrective procedures should be reserved for deformities of over three years' standing, since slight curvatures usually respond to nonoperative antirachitic measures.

As auxiliaries, especially with the view of overcoming the anemia and the deficiency of mineral elements, the syrupus calcii et sodii hypophosphitum (N. F.), the syrupus hypophosphitum compositus (U.

S. P.), the syrupus ferri iodidi and the liquor phosphori (N. F.) are of
great therapeutic value. However, cod liver oil is the *specific* in rachitis
and may advantageously be combined with the aforementioned remedies.
(See p. 451.)

In intractable cases organotherapy, especially the extracts of thy-
roid, thymus, and pituitary glands and red bone marrow should be
given a fair trial. A sojourn at the seashore is highly to be recom-
mended. Cases of florid rachitis of older children up to ten years of
age are on record. They are usually spoken of as rachitis tarda.

Achondroplasia*

(CHONDRODYSTROPHIA FETALIS, FETAL RICKETS, MICROMELIA)

These terms are used to designate a peculiar type of congenital
dwarfism arising from early fetal arrest of growth of the bones that

Fig. 148.—Achondroplasia in ten-month-old baby. Note great length of trunk as
compared with the short extremities.

*Though not an acquired disease, this subject is treated here in order to emphasize its
many differences from rickets.

are formed in cartilage, leaving the bones that are laid down in membrane unaffected. Thus, we have shortening of the extremities, and of the bones of the base of the skull, while the bones of the vault of the cranium and the trunk are normal. This peculiar chondral dystrophy produces the following characteristic statural disparities:

Fig. 149.—Achondroplasia (left). Both children are of the same age. Note the short legs and long trunk in the achondroplasiac as compared with the normal (right) child. (After Drs. Wood and Hewlett.)

Shortness of the extremities as compared with the normal (relatively long) abdomen; the forearms are longer than the arms and the legs longer than the thighs; bowing of the extremities, especially lower, and thickening of the terminal epiphyses; limited power of extension of the upper extremities; peculiar fan-like divergence of the thick, uni-

formly sized fingers, the so-called "trident hand"; marked narrowing of the pelvis; lordosis; protuberant abdomen; narrowing of the base of the skull ("pug-nose," broadening of the jaws) as compared with the normal (relatively large) upper part of the skull. The skin and nails are normal; the hair is soft and abundant in growth. Intellect is usually fairly normal. The great majority of cases of achondroplasia die in utero or soon after birth. Those who survive may attain old age. They very rarely exceed 4 feet in height.

Scorbutus Infantum

(Moeller-Barlow's Disease, Scurvy, Acute Rickets)

Infantile scurvy is an acute specific hemorrhagic affection of as yet unknown origin. It is probably due to direct microbic infection or toxemia resulting from intestinal putrefaction. As the disease occurs principally in infants from six to eighteen months old, the period when nutritional disturbances are most rampant, there is every reason to believe that malnutrition is the most active predisposing cause. This explains also the frequency with which infantile scurvy is observed in infants fed exclusively on boiled or sterilized milk (milk deprived of some of its nutritious qualities), or poor breast milk, in short, on food lacking some essential elements (vitamines*). I had the opportunity to observe scurvy in a pair of twins six and a half months old, who were partly breast fed. The disease developed in both of them almost at the same time, and subsided promptly on the administration of small quantities of lemon juice, mixed vegetable soups and raw milk in addition to the breast feeding. The simultaneous occurrence of the affection in both babies and the absence of a history of faulty feeding tend to the infectious theory of the causation of scurvy. The principal pathologic changes in scurvy consist of an increase in the width and vascularization of the cartilage zone and hemorrhage into the loose vascular layer of the connective tissue of the periosteum adjacent to the bone, thus leading to detachment of the periosteum from the bone and forming a thick sheath of blood clot underneath it. The lower and upper extremities and the ribs are most frequently affected. Hemorrhages take place also in the mucous membranes of the hard palate and gums, in the muscles and more rarely in the serous cavities and solid viscera. There is anemia but no leucocytosis. The calcium content of the blood is decidedly diminished (Hess).

The onset of the disease is usually sudden or, less frequently, preceded by malaise or digestive disturbance of a few days' duration

and slight fever. The child is restless, cries when it tries to move itself or when it is being handled. This symptom is the result of pain and tenderness especially in the lower extremities. For fear of pain the patient instinctively ceases to move its limbs (pseudoparalysis). Examination of the extremities soon reveals at the diaphyses of one or both femurs, more rarely of the tibia and fibula, or upper limbs, spindle-shaped, colorless, smooth, nonfluctuating swellings surrounding the bones. The tumefactions for the most part are due to subperiosteal hemorrhage. Exceptionally there is bleeding also from

Fig. 150.—Scorbutus in a fifteen-months-old infant. Note hemorrhage from the gums and in the skin, and swelling of lower extremities.

beneath the periosteum of the ribs and of the bones of the head (protrusion of the eyeball in subperiosteal hemorrhage of the frontal bone) and face, and occasionally spontaneous separation of the epiphysis from the shaft of the bone, leading to bone infraction, impaction or fracture. The next important symptom of infantile scurvy is sponginess and discoloration (minute transient ecchymoses) of the gums,

with a tendency to bleed. In quite a number of cases the hemorrhagic tendency extends also to the skin, subcutaneous tissue (typical "black eye" after a fit of crying or laughing, also discoloration and proptosis of an eye resembling that of chloroma), to the mucous membranes and the viscera (dysentery!), so that as a result of loss of blood profound anemia, edema and albuminuria supervene. On the other hand, some cases pursue a very mild course (*formes frustes*), especially if recognized early and treated energetically. Except occasional permanent hyperostosis of the affected shafts of the extremities, the prognosis as a whole is favorable, recovery usually taking place within from a few weeks to as many months. Neglected cases, however, may end fatally from the aforementioned complications, or from pneumonia.

Treatment.—An antiscorbutic diet and fresh air form the treatment *par excellence*. Prompt improvement and rapid recovery usually follow the administration of fresh cow's milk, fresh fruit juice (lemon, orange, or pineapple), fresh vegetable soups, beef juice, and in older children fresh eggs and vegetables (potato puree, carrots, tomatoes, fresh or canned—Hess—spinach, etc.). Where convalescence is protracted we may prescribe the compound syrup of hypophosphites (U.S.P.) with extract of malt and cod liver oil.

Infantile scurvy may be mistaken for rheumatism, peliosis rheumatica, purpura hemorrhagica, syphilitic epiphysitis, osteomyelitis, rickets, and occasionally (when the orbit is involved) for chloroma.

In *rheumatism* the swelling is usually localized at the articulations and "jumps" from one place to another. It is accompanied by fever and responds to the salicylates. Hemorrhages are absent.

Peliosis rheumatica is characterized by deep red or bluish spots, as a rule, limited to the extremities. It usually occurs in older children.

Purpura hemorrhagica is free from diaphyseal hematomas and pain.

Syphilitic epiphysitis is free from the hemorrhagic tendency, and often presents other syphilitic lesions.

Osteomyelitis is associated with high fever and rapid local suppuration.

Rickets is free from acute pain and hemorrhagic symptoms, but has other pathognomonic symptoms. It responds very slowly to treatment. It is worth remembering, however, that rickets and scurvy may coexist.

Chloroma or green tumor usually shows a predilection for the skull (temporal fossæ and orbits), giving the child a characteristic frog-like

appearance. It is a grave blood disease—profound anemia with relative and absolute increase in lymphocytes.

Beriberi

(Kakké. Polyneuritis Endemica)

There is still considerable diversity of opinion regarding the etiology of beriberi. While the majority of observers attribute the disease to a polished-rice-diet, to an insufficiency in vitamines,[†] some clinicians believe it to be due to an unknown infectious agent. Beriberi prevails extensively in Japan, China, Indo-China, Borneo, Philippine Islands, Straits Settlements, Malay States, Java and Sumatra, Brazil and Iceland..

It ordinarily runs an afebrile course, developing insidiously with epigastric pain, debility, sensation of precordial oppression and other symptoms of dilatation of the heart. Physical examination elicits, weakness of the extremities, hobbling gait with the legs widely apart: immobility to stand with the eyes shut, wasting of the anterior tibial and peroneal muscles, loss of knee jerks, preceded by exaggeration; later also wrist drop, and edema, especially of the lower extremities, in the absence of other signs of nephritis. Sometimes the disease runs an acute course with paralysis spreading to the respiratory muscles, when it usually proves fatal. Otherwise the mortality ranges between from 5 to 50 per cent, all depending upon how early the treatment is initiated.

Treatment.—Rest in bed, nutritious diet (fresh milk, fresh fruit and and vegetables, meat broths and juice, unpolished rice and other cereals), autolyzed yeast[*] (5 to 30 drops t. i. d.), tonics, such as iron, quinine and strychnine and sometimes digitalis in cases of heart weakness.

Pellagra

This disease is not rarely met with in this country, especially in the south. Its cause is still the subject of considerable controversy. Some clinicians[1] attribute it to a vegetarian diet,[†] with a preponderance of cereals, others[2] maintain that it is an insect-borne infection, since it prevails during the summer and early autumn, when certain gnats of the genus Simulium abound. It may readily be assumed that while

---

[†]See page 114.

[*]Dry pressed brewer's yeast is placed in an incubator at a temperature of 37.5° C. for 32 hours; the liquid is allowed to gravitate through a paper filter and then kept at room temperature for another 10 hours until the purin bodies have separated when it is again filtered. The autolyzed yeast if kept on ice will not spoil for quite a long time. It is not to be used if mould forms.

[1]Goldberger, Wheeler and Lydenstricker (Jour. A. M. A., September 21, 1918).

[2]Jobbling and Petersen (Jour. Infect. Dis., Vol. 18).

an infection is the exciting cause of the disease, a deficiency of fresh green vegetables and animal protein in the dietary serves as the most important predisposing cause.

Pellagra is characterized by symmetrical erythema or dermatitis, chiefly on the exposed surfaces (neck, face and extremities), red, fissured tongue, diarrhea or constipation, restlessness, insomnia, paresthesia, and disturbance of the knee jerks (either exaggerated or absent).

It is readily curable by change of climate, restriction of cereals, and feeding on animal proteins, such as fresh milk, beef juice, broths, eggs, meats, etc., also fresh fruits. Hematic tonics. The skin should be protected from the rays of the sun, and the eruptions treated with calamine lotion and similar mild remedies.

Diabetes Mellitus

(Glycosuria)

Within recent years, with increased interest in accurate diagnosis, the number of cases of diabetes in children recorded has greatly increased. In former years undoubtedly many of the rapidly fatal cases escaped observation. The importance of careful examination of the urine of older children and infants suffering from polyuria or enuresis, therefore, cannot too strongly be emphasized.

We distinguish two forms of glycosuria: glycosuria spuria (temporary or dietetic), and glycosuria vera (diabetes mellitus). The first variety is comparatively of little clinical importance. It is the result of consumption of sugar greater in quantity than can be assimilated, and usually disappears after arrest of the causal factor.

On the other hand, *diabetes mellitus* is an extremely fatal affection, death taking place, in violent cases, sometimes after a few weeks or months, or in less acute cases, often within a year or two at the latest. This variety is often hereditary.

The onset of diabetes mellitus is rather sudden. The child begins rapidly to lose in weight, notwithstanding good appetite, suffers from excessive thirst, passes a large quantity (75 to 115 ounces) of urine (often enuresis nocturna as well as diurna!), of high specific gravity (1,030), containing a large proportion of sugar, at times acetone and diacetic acid, and loses in vitality from day to day. In addition to these symptoms there are also digestive disturbances, dryness of the skin, skin affections (furunculosis, lichen-like eruption with severe itching) onychitis, cataract, nerve disorders (*e. g.*, Friedreich's ataxia), obstinate acetone odor from the mouth, etc. The course of the disease varies. As a rule, it

is more rapid than in adults; the younger the patient the more violent the course. Death usually occurs as a result of general exhaustion or intercurrent diseases, such as pneumonia, tuberculosis, and the like, and is frequently preceded by coma diabeticum or uremia. Recoveries, however, are also on record.

Treatment.—Every effort should be made to trace the cause of the disease and to combat it energetically. As congenital or acquired syphilis has frequently been found to play an essential part in the causation of diabetes, it is prudent to subject the patient to a course of antisyphilitic treatment. We have no means at our command to influence the other supposed etiologic factors of diabetes, such as traumatism to the head, shock, tuberculosis, various infectious diseases, etc. The time is not distant, however, when the true nature of the affection will be disclosed, and the remedies found which will greatly aid us in the prevention and arrest of the disease at its very inception. Until this blissful moment arrives we will have to continue groping in the dark, empirically treat symptoms, and depend chiefly upon a restricted diet, which at best never strikes the root of the evil, and is hardly practicable in diabetes of early childhood. Wherever possible (especially in older children), the diet should consist of fresh meat soups and broths; bread and biscuits of gluten flour, with cream and butter; eggs; moderate quantities of meats of all

FOODS ARRANGED ACCORDING TO THEIR APPROXIMATE PERCENTAGE OF CARBOHYDRATES (DR. HALPERN)

(1) 5%

Fresh Vegetables:

Lettuce	Tomatoes
Spinach	Rhubarb
Sauerkraut	Leeks
String beans	
Celery	Egg plant
Asparagus	Cabbage
Cucumbers	Radishes
Brussels sprouts	
Sorrel	Beet greens
Endive	Water cress
Dandelion greens	
Swiss chard	
	Pumpkin
Sea kale	Kohl-rabi
Broccoli	
Vegetable marrow	
Cauliflower	

Canned Vegetables:

Asparagus
Spinach
String beans

Fruits:

Ripe olives
Grape fruit

Nuts:

Butternuts
Pignolias

Miscellaneous:

Unsweetened & unspiced pickles,—clams, fish, oysters, scallops, liver, roe.

(2) 10%

Fresh Vegetables:

Onions	Carrots
Squash	Okra
Turnips	Beets
Mushrooms	

Fruits:

Lemons
Oranges
Cranberries
Strawberries
Blackberries

Gooseberries
Peaches
Pineapple
Watermelon

Nuts:

Brazil
Black walnuts
Hickory
Pecan
Filbert

(3) 15%

Fresh Vegetables:

Green peas
Artichokes
Parsnips

Canned Vegetables:

Lima beans

Fruits:

Apples
Pears
Apricots
Cherries
Blueberries

Currants
Raspberries
Huckleberries

Nuts:

Almonds
English walnuts
Beechnuts
Pistachio
Pine

(4) 20%

Fresh Vegetables:

Potatoes
Shell beans
Baked beans
Green corn
Boiled rice
Boiled macaroni

Fruits:

Plums	Bananas

Nuts:

Peanuts

(5) 40%

Chestnuts

kinds, and fish, oysters and scallops; well boiled spinach, asparagus, string beans, cauliflower, cabbage, radishes, and turnips; protein milk; fresh sour fruit, such as grapefruit, lemon, occasionally cranberries and blackberries. Saccharin instead of sugar. In infants milk and amylacea are indispensable, but should be restricted as much as possible. Oatmeal gruel seems to work well in some cases. Mild hydrotherapeutic procedures, and light exercise are useful. Bicarbonate of soda in large doses should be administered to prevent acidosis. Opium, in some form, and arsenic, in addition to cod liver oil and iron, are the only drugs of therapeutic value. Complications should be treated according to indications. Koplik is of the opinion that the Allen treatment of fasting, as employed in adults, is also applicable in children. During the treatment the child should be kept in bed.

Diabetes Insipidus

(Polyuria)

Polyuria, like glycosuria, may be transient or persistent. Transient polyuria is quite common in children and is usually of nervous origin. On the other hand, persistent polyuria—diabetes insipidus—is comparatively rare. It is manifested by excessive thirst, polyuria (pale, sugar-free urine of low specific gravity, not exceeding 1,006), dry skin, disturbances of the digestive and nervous systems. The course is very protracted, but the prognosis *quoad vitam* favorable. Permanent recovery is rare.

As the etiology is obscure (disease of the hypophysis cerebri?), little can be expected from treatment except in cases due to syphilis which frequently yield to antisyphilitic medication. Change of air, hydrotherapy, a nitrogenous diet and an ample supply of water act beneficially.

A number of clinicians have lately been recommending pituitary solution (posterior lobe) in doses of from 0.25 to 1 c.c. subcutaneously and also by mouth. The output of urine is reduced, but only temporarily.

Adipositas

(Lipomatosis Universalis, Obesity)

Contrary to what is observed in older children or adults, overfatness in infants very rarely gives rise to constitutional disturbances. As a rule, the fatness subsides when the child begins to walk about.

In older children obesity is often associated with marked anemia, shortness of breath and fatty degeneration of the heart. If such symp-

Fig. 151.—Adipositas; child weighs thirty-six pounds at eight months.

toms appear, it is essential to eliminate fats and carbohydrates from the dietary and to recommend systematic exercise, active massage, and hydropathic procedures. Carlsbad salts and thyroid gland substance are often useful; some cases, however, resist all sorts of treatment, and readily succumb to intercurrent diseases.

Adipositas should not be mistaken for cretinism (*q. v.*) and Frölich's Syndrome (*q. v.*).

Exudative Diathesis

This symptom complex, first fully elucidated by Czerny, is quite frequently observed in infants of certain predisposed families. It does not seem to be congenital in nature although some clinicians claim to recognize the diathesis in the newborn by a prominent comb-shaped tuft of hair in the centre of the scalp. The affection is characterized by the combination of inflammatory symptoms of the skin and mucous membranes, as follows: (1) The skin: transient erythema, intertrigo, urticaria, prurigo, blepharitis, phlyctenular conjunctivitis, and seborrheic eczema; (2) the respiratory tract; recurrent angina, pharyngitis, coryza, laryngitis diffuse bronchitis and tendency to asthma; (3) the alimentary tract: stomatitis, lingua geographica, unprovoked diar-

rhea and mucous colitis. Excepting the presence of marked eosinophi-
lia (from 10 to 20 per cent), the blood shows no definite alterations.
The nervous system is but slightly involved (vasomotor disturbance,
as manifested by transient flushing of the face). Some authors attrib-
ute pavor nocturnus, spasmophilia and incontinentia urinæ to the
exudative diathesis. The general appearance of the baby may vary
from puniness to obesity, in either case accompanied by muscular
atony and general lymphatic enlargement.

Czerny attributes the condition to faulty food assimilation, partic-
ularly of fat, giving rise to endogenous nutritional noxa and conse-
quent increased susceptibility to local infections, and anaphylaxis.
It is also caused by ectogenic nutritional noxa, resulting from over-
feeding by a rich diet, be it proteins, fats or carbohydrates. In some
cases the food idiosyncrasy may be determined by the "Allergy test."
(See p. 87.)

Treatment.—This must be directed principally to the suitable se-
lection of the dietary. The supply of milk, even to the very young
infant, must be limited to the bare necessity of life, paying particular
attention to elimination of fat. The milk at all times should be diluted
with cereal gruels. Older babies should be fed on cereals, well-boiled
vegetables and bread, and but little milk. Fruit, raw or cooked, is al-
lowed. Careful attention should be paid to the nose and throat, and to
the skin. Outdoor air, preferably in the country.

Acidosis

(RECURRENT, CYCLIC, PERIODIC, VOMITING. "ACID INTOXICATION")

Cautley defines acidosis as "an abnormal metabolism of carbon, lead-
ing to the appearance of organic acids in the blood and urine, and the
formation of ammonia to neutralize the acids." The chief evidences of
acidosis are the presence of acetone bodies (acetone, diacetic acid,
beta-oxybutyric acid) in the urine, diminished alkalinity of the blood
(as readily determined by the phenolsulphonephthalein test) and reduced
CO_2 tension* in the alveolar air (tested by the Plesch-Howland appara-
tus). It is well to remember, however, that the presence of acetone bodies
in the urine is not pathognomonic of acidosis alone, since they are not
rarely observed in acute febrile diseases, starvation and cachexia, acute
yellow atrophy of the liver, and delayed anesthesia poisoning. The
cause of acidosis is still awaiting definite solution. Ewing suggests
that acidosis is due to disturbance of fat metabolism, caused by de-

*Normal tension is about 45 mm. Hg., equaling 6 per cent CO_2; anything below 30 mm. is an
indication of acidosis.

ficient hepatic function associated with the absorption of alimentary toxins. Mellanby believes acidosis to be caused by derangement of the glycogen function of the liver, leading to imperfect metabolism of the fats with formation of the aforementioned acids as intermediary products and imperfect protein metabolism and creatin formation due to carbohydrate insufficiency. In view of the fact that acidosis has occasionally been met in epidemic form, some authors are inclined to attribute it to an obscure systemic infection or a species of toxemia. According to C. H. Dunn, acidosis prevailed around Boston in the winter of 1915 and 1916, but he thinks it was symptomatic of a grip-like infection of the upper air passages.

Acidosis is most common in children from two to five years old. It is manifested clinically by sudden attacks of vomiting, anuria, prostration, sopor, and hyperpnea. The respirations may reach up to sixty per minute, yet be free from dyspnea and cyanosis (the patient's lips are usually deep red in color). The vomiting recurs, periodically, cyclically, at short or long intervals, is incessant and uncontrollable, often blood and bile stained, and occasionally so intense as to produce alarming hemorrhage from the stomach. The vomitus in the beginning has a "sweetish" odor. The attacks may last from a few hours to several days, and abruptly end in perfect recovery of the patient or, exceptionally, lead to a fatal issue, particularly if not properly handled. The temperature is moderate (except when the acidosis is complicated by pyelitis which is not rarely the case especially in girls); the pulse at first somewhat retarded, and the blood shows a marked leucocytosis. Often the lips are bright red.

With these symptoms in view there ought to be no difficulty to arrive at a correct diagnosis. It may, however, be mistaken for appendicitis, recurrent uremia from chronic nephritis, and tuberculous meningitis, all of which diseases of course have pathognomonic symptoms of their own.

Treatment.—After brisk catharsis (calomel gr. ii), stop all liquids by mouth. An attempt may be made to give bicarbonate of soda in 10 grain-doses (by putting it dry on the tongue and letting it melt in the mouth) every hour or two, to counteract the acidity, but if it is promptly rejected, it is best left alone, and administered by rectum instead (1 ounce of bicarbonate of soda in 1 pint of warm water). The irrigation should be repeated every four hours and followed half an hour later by nutrient enemas consisting of a 5 per cent dextrose solution, 4 ounces at a time. If dextrose is not obtainable, saccharose may be used instead. Occasionally, we may succeed in arresting the vomiting by administering $\frac{1}{16}$ grain of codeine sulphate (without water)

every four hours. Hot moist packs to induce diaphoresis often act beneficially. After the vomiting has ceased for about twelve hours, we may begin feeding by mouth, giving a teaspoonful to a tablespoonful of the dextrose solution every two hours; later zwieback, soda biscuits, or toast, cereals with small quantities of skimmed milk, and gradually resume the regular diet. During the intervals between the attacks, overfeeding should be avoided and the use of fats restricted.

Test for Acetone and Diacetic Acid.—Test solution consists of 10 grams glacial acetic and 10 c.c. of 1:10 solution of sodium nitroprusside. Add 20 drops of this reagent to 15 c.c. of filtered urine in a test tube. Overlay the mixture with ammonia water. The presence of acetone, even 1 part in 2,000, causes a purple ring at the surface, separating the two fluids. Dilute urine with 4 parts of water, add drop by drop solution of ferric chloride diluted 1:10. Normal urine or that containing acetone will show a cloudy white precipitate. Presence of diacetic acid, even 1/10,000, gives a purplish black cloudy precipitate.

DISEASES OF THE CIRCULATORY SYSTEM

CONGENITAL HEART DISEASE

(Vitium Cordis)

As a rule, infants born with heart disease are very delicate. Most of them are born asphyxiated and if resuscitated remain cyanotic,[1] or very anemic, atelectatic, cry feebly, breathe superficially and very rap-

Fig. 152.—Vitium cordis. "Morbus cœruleus." Note "club-shaped" fingers and cyanosis (represented by dark patches on face and lips), in a child eight years old.

---

[1]From time immemorial cyanosis (morbus coeruleus or "blue sickness") has been looked upon as a cardinal symptom of congenital heart disease. It is usually associated with clubbing of the fingers and toes. Its diagnostic importance has been greatly exaggerated, since it is not rarely absent in the severest forms of congenital vitium cordis.

idly, are barely able to suckle, present a very weak pulse and subnormal temperature. Not rarely they are born prematurely* and with congenital defects of other parts of the body. Some children present a club-shaped appearance of the fingers and toes at an early age; some of them later. If they survive for any length of time, their growth and development are very much delayed. They are helpless, begin to hold up the head or sit up at a much later age than the normal baby. When they start to walk they tire very rapidly. They rarely creep and when on the floor are often unable to lift themselves. They are very susceptible to colds, and once taken sick, they are very slow to recuperate. Bottle-fed babies frequently succumb to gastrointestinal diseases, even of comparatively simple nature. If they live up to school age and are more frequently exposed to acute contagious and infectious diseases, their weakened constitution forms a favorable nidus for the contraction of these affections, and is rarely able to withstand them.

Even under the best of care, children with congenital heart disease usually live but a few years. Death sometimes occurs suddenly, or incidentally in the course of other diseases which in normal children are not dangerous to life, especially respiratory affections. Unless the heart defect is very mild in nature, children with vitium cordis rarely survive the age of puberty.

The course of congenital heart disease varies, of course, with the severity of the defect, but practically resembles that of acquired vitium cordis, which is fully described in other parts of this treatise. The following are the most common congenital heart affections.

Persistence of the Foramen Ovale

This condition is the result either of faulty construction of the foramen or its valves, or defects in other portions of the heart (e. g., stenosis of the pulmonary artery) which by indirect blood pressure prevent complete obliteration of the foramen.

It is the most frequent kind of congenital heart disease, but is not always recognizable during life. In the presence of clinical symptoms the diagnosis may be based upon preponderance of cyanosis, a systolic blowing sound at the base of the heart or over the third or fourth costal cartilage.

Persistence of the Ductus Arteriosus Botalli

Complete obliteration of this duct is supposed to occur by the end of the third month. This may be retarded or may entirely fail—

---

*See "Feeble Vitality of the Newborn," p. 213.

usually in cases where the left ventricle is not properly filled with each heart cycle (*e. g.*, in atelectasis, fetal pneumonia, stenosis of the pulmonary artery), in which event the blood from the pulmonary artery continues to flow through the ductus arteriosus to the insufficiently filled aorta. As a result of this anomaly there develops sooner or later hypertrophy of the right ventricle usually with dilatation of the pulmonary artery.

The symptomatology is very variable. In cases of only partial patency the symptoms may be so slight as to escape observation. Complete patency of the duct very gradually gives rise to the following group of symptoms: disposition to respiratory affections, cyanosis, or waxy pallor, dyspnea, cool extremities, palpitation, a thrill over the anterior chest wall, increased cardiac dulness to the right, accentuation of the second pulmonic sound which can also be heard in the carotids, loud, buzzing, systolic murmur over the precordium, often epistaxis or hemorrhage from other mucous membranes, and finally, sometimes not until after several years of existence, marked symptoms of failure of compensation with rapid fatal determination.

Defects in the Septum Ventriculorum

(COMMUNICATION OF THE VENTRICLES)

It is a very common condition, most frequently the result of fetal myocardial diseased processes, and not rarely coexisting with congenital stenosis of the pulmonary artery. The defect is situated either in the anterior or posterior portion of the septum. Very rarely the whole wall between the ventricles and auricles is absent, so that all four heart cavities communicate.

Accentuation of the second pulmonic sound; overfilling of the veins; marked cyanosis developing soon after birth or, more gradually, sometime after; and hypertrophy and dilatation of the right ventricle, all point to a defect of the ventricular septum. A positive diagnosis, however, is almost impossible during the life of the patient.

The prognosis is very bad.

Congenital Stenosis of the Pulmonary Artery

The stenosis may involve the orifice alone, the entire trunk, or the branches of the pulmonary artery. Accordingly the symptomatology varies with the extent and location of the lesion. As a rule, there is marked cyanosis from birth. Some children are born asphyxiated, and if resuscitated, continue to suffer from attacks of suffocation and convulsions, to which they usually succumb within the first few days

of life. Stronger children may survive these attacks, gain some
strength, lose part of the cyanosis and live several years.

Physical examination reveals arching of the anterior left chest wall,
enlargement of the cardiac area, chiefly to the right, a diffuse systolic
murmur, heard loudest over the left and third costal cartilages, and
often a purring thrill on palpation. The blood usually shows a marked
increase in the number of erythrocytes and a high hemoglobin index.

Congenital Stenosis of the Tricuspid Valve

It is usually the result of an anomalous or excessive development of
the muscle substance of the valve, or of fetal endocarditis, and is often
associated with other congenital heart defects.

The symptomatology resembles that of defects of the pulmonary
artery, except that the murmur is heard loudest over the fourth and
fifth costal cartilages, and hypertrophy of the right side is either
absent or very slight.

The prognosis is unfavorable.

Congenital Stenosis of the Ostium Atrioventriculare Sinistrum

(Stenosis of the Aorta)

The stenosis may be situated at the point of origin of the aorta; at
any place throughout the entire aortic system; or at the ductus Botalli.

As a result of either one of the aforementioned conditions there is
hypertrophy of the left heart. Varying with the seat of the atresia,
the blood vessels above the lesion may be abnormally filled with blood,
while those emerging below the lesion suffer from a deficiency of it.
Between the two groups of vessels a collateral circulation is usually
established which may frequently be recognized by numerous, visible,
actively pulsating, subcutaneous blood vessels over the thorax. A sys-
tolic murmur is often heard over the dilated arteries. The heart is
usually free from any auscultatory signs, unless the orifice of the aorta
be involved, when a loud systolic murmur may be heard at midsternum.

The patient may live for several years—until compensation rup-
tures. Death sometimes ensues very suddenly from rupture of a group
of vessels above the stenosis.

Treatment.—The treatment of congenital heart disease is practically
the same as that of acquired and is fully outlined on p. 540. Complete
rest in the strictest sense of the word will help to prolong life—possi-
ly to an advanced age.

Dextrocardia

Among the few congenital malpositions of the heart (*mesocardia*—the heart occupies a central position of the chest wall; *ectopia cordis*—the heart may be situated either between a fissure in the sternum immediately beneath the skin, in the neck or in the abdomen below the diaphragm), *dextrocardia* (see Fig. 153), or transposition of the heart to the right side, is of special interest inasmuch as it very rarely interferes with the life or welfare of the patient. Dextrocardia is often associated with a general transposition of the viscera. The aorta and

Fig. 153.—Dextrocardia in a girl six years old. Posterior view.

its branches usually remain in their normal situation. Dextrocardia should not be confounded with displacement of the heart by large effusions or growths in the thoracic cavity.

ACQUIRED HEART DISEASE

Myocarditis

Degeneration of the muscular tissue of the heart is occasionally congenital, a sequel of infection during fetal life, but most frequently acquired, occurring either secondarily to acute infectious diseases, or

as a result of extension of an inflammation of the inner or outer lining of the heart.

The inflammation may be diffuse or circumscribed, and as in adults, either plastic or interstitial, or degenerative or parenchymatous.

The interstitial variety of myocarditis usually leads to suppuration and abscess formation of the musculature. In parenchymatous myocarditis the transverse striæ of the fibrillæ appear lost, the muscle consisting chiefly of fatty and granular substances.

The course of the disease varies greatly with the underlying cause and the rapidity of the inflammatory process.

In the majority of instances *interstitial* myocarditis is complicated by endocarditis and pericarditis, and hence it is very seldom possible early to diagnose the existence of the myocarditis. In cases where the inflammation is circumscribed, myocarditis may be surmised by the sudden precordial pain, dyspnea, high fever, restlessness and delirium. The apex beat and pulse are weak, arrhythmic and rapid. Death is the usual termination; not rarely occurring suddenly with symptoms of sudden collapse.

Parenchymatous myocarditis ordinarily runs a slow and latent course. Occasionally, however, the degenerative process develops quite rapidly. Extreme pallor, breathlessness, and weak and galloping pulse point to the involvement of the myocardium, but in the early stages the diagnosis can rarely be made with any degree of certainty. As the disease advances and symptoms of cardiac dilatation and passive pulmonary congestion set in, the diagnosis is fairly certain.

The treatment is the same as in endocarditis (*q. v.*)

Pericarditis

Primary pericarditis is usually due to a streptococcus or pneumococcus infection through the blood or lymph channels or in connection with acute articular rheumatism. Like pleuritis, inflammation of the pericardium may occur in dry form or with an effusion. The exudation may be serofibrinous, hemorrhagic, or purulent. Dry, as well as exudative, pericarditis may give rise to inflammatory adhesions between the pericardium and the heart, and occasionally to the anterior and posterior chest walls and vertebral column.

The gravity of this affection should, therefore, not be underestimated. The prognosis is serious, especially in the secondary variety occurring in connection with tuberculosis, septic processes, pleuropneumonia, caries of ribs or vertebræ, severe exanthematous diseases (*e. g.*, scarlatina), purpura hemorrhagica, chronic nephritis, etc. It is less dangerous in primary, usually rheumatic form, particularly if the

patient is over three years of age, or when caused by syphilis and is detected and treated early.

Bearing in mind the etiologic factors just enumerated, we can readily appreciate that pericarditis in children must be quite common. Indeed, there is ample reason for the belief that in children over three years of age pericarditis is almost as frequent as endocarditis, with which affection, by the way, it is not rarely associated.

The onset of primary pericarditis is usually very sudden, but sometimes, like the secondary variety, it may be insidious. Ordinarily it is ushered in with high temperature, vomiting, cardiac oppression, orthopnea, dyspnea, and accelerated pulse. Cough is an early symptom, and, in the presence of an effusion, quite pronounced. This symptom is probably due to cardiac pressure against the lungs. The pulse, which in dry pericarditis is strong, is often very feeble, barely perceptible, and irregular in marked exudative pericarditis. Pain is frequently intense, especially if associated with polyarthritis. The patient is restless, sleepless, the expression of his face anxious, and denoting great suffering. Of course, the symptomatology is greatly modified by that of the underlying affection, if existing.

The physical signs vary with the stage of the disease. Before the development of the effusion, auscultation elicits superficial, exocardial, to-and-fro friction and creaking sounds, limited over the cardiac region, often changeable with the position of the patient and audible independently of the heart sounds. Friction fremitus may be felt over the area where the friction murmur is heard. Endocardial murmurs may coexist. When serous effusion occurs the friction sound is found diminished or absent, the heart impulse very feeble, whereas the pulse may be felt quite strong, and the respiratory movements of the left side of the chest are diminished. The area of heart dulness is greatly increased laterally and vertically, pushing the edges of the lungs aside so that the entire sternal region is dull on percussion. When the effusion is large, we can also note distinct bulging of the cardiac area of the chest. According to Rotch, the liver is depressed and a dull note is obtained in the right fifth intercostal space. He considers this a sign of great importance in the differentiation of pericardial effusion from cardiac dilatation, since in a dilated heart the dulness, he thinks, never reaches the fifth interspace.

There are several other distinctive features which render the differentiation of pericardial effusion from enlarged heart possible. Thus, in dilatation or hypertrophy of the left ventricle, the apex beat is felt at the extreme left limit of the dulness (outside the mammary line) and at its lowest level, while in effusion the apex beat, or rather the

heart impulse, is at a spot inside and above the boundaries of the cardiac dulness, somewhere between the fourth and third interspace. In pericarditis the dulness develops much more acutely than in an enlarged heart, which latter occurs secondarily to more or less chronic valvular disease. However, we should bear in mind that pericarditis, acute or chronic endocarditis, and hypertrophy and dilatation may co-exist and give rise to a symptom complex beyond the possibility of individualization. For further differentiation between pericarditis and endocarditis the reader is referred to the discussion of the latter affection. (See p. 536.)

With absorption of the fluid in the pericardium there is a gradual return of the symptoms of the first stage and, in favorable cases, *restitutio ad integrum,* or quite frequently, supervention of pericardial adhesions with consecutive systolic retraction of the chest wall over the entire precordium.

The nature of the effusion can readily be ascertained by exploratory puncture, but even without it we may surmise the presence of *pus* if the pericarditis develops secondarily to septic processes; *blood,* after severe trauma, and *serum,* in primary, usually rheumatic, pericarditis. An x-ray examination is often of service. The determination of the character of the effusion is important especially as regards the further course and treatment of the disease.

Rheumatic pericarditis, if free from complications, lasts from two to three weeks or longer. After about ten days there is a gradual evanescence of the symptoms. Not infrequently, however, the apparent recovery is only temporary, inasmuch as there may be a return of the effusion, or development of valvular deposits, which sooner or later give rise to marked valvular disease. These manifestations are particularly prone to occur in pericarditis with polyarthritis. Pericarditis, like endocarditis, not rarely precedes the joint symptoms, may run a latent course and if mild in character disappear again without being detected, possibly not until repeated recurrences and appearance of complications. More rarely, pericarditis ends in death either rapidly as a result of cardiac muscular insufficiency and pulmonary edema, or more slowly from early complications, such as pleurisy, pneumonia, severe adhesions, endocarditis, etc.

Purulent pericarditis pursues a much more violent course. Extreme fatigue, severe attacks of syncope and pyemic fever predominate, while the local symptoms are comparatively insignificant. Even the exudation is often slight. When it occurs in conjunction with tuberculosis, it is very malignant in character. It is then manifested by enormous hypertrophy of the pericardium, extensive adhesions, large quantities

of pus between the heart and pericardium, and numerous tubercles in the latter. It is invariably fatal. The same holds true for pyemic pericarditis, in which streptococci, pneumococci, staphylococci and less frequently, gonococci act as the principal exciting cause.

Treatment.—A disease presenting so many phases as pericarditis, can at best be treated only symptomatically. Absolute rest in bed, liquid diet (thin cereals, vegetable soups, fermented milk), an ice bag (not too heavy and preferably on top of a layer of lint), to the precordium, and sodium salicylate (1 grain for every year of the child's age every two hours) and codeine ($\frac{1}{30}$ grain every six hours) internally will often do well in rheumatic cases. In large pericardial serous effusions with threatening syncope we may try free diuresis and saline catharsis with or without aspiration (in the fifth intercostal space a little to the left of the border of the sternum). The latter procedure frequently proves useful also in small nontuberculous purulent effusions, while in large purulent effusions incision and drainage are preferable to aspiration. In these cases some benefit may be derived from vaccines.

In quite a number of cases sodium iodide in from 3 to 5 grain doses, t.i.d., seems to exert a specific effect; and, bearing in mind also the possibility of underlying syphilis, we should always administer this remedy irrespective of the variety of the pericarditis and the mode of treatment simultaneously employed. Digitalis or strophanthus may be given to strengthen the heart, if cardiac weakness sets in, which is apt to occur later in the course of the disease.

Chronic pericarditis is productive of grave disturbances of the circulation, cardiac hypertrophy, and dilatation. Myocarditis is a frequent sequela. See also "Congestive Cirrhosis" and "Sugar-Cake Liver".

Endocarditis Acuta

The etiologic factors of acute endocarditis are essentially the same as in pericarditis (*q. v.*) except that the former is more frequently associated with rheumatic affections, such as arthritis, chorea, tonsillitis, erythema nodosum, etc., and not rarely complicates pericarditis. Invasion of the endocardium by the streptococcus, staphylococcus, pneumococcus, the bacillus pyocyaneus, tubercle bacillus, and gonococcus usually occurs through the circulating blood, giving rise to a pathologic condition very similar to that observed in adults.

The inflammation, which is usually limited to the left side of the heart (in the fetus the right side is mostly affected), first attacks the vascular layer of the endocardium between the muscular and fibrous coats, resulting in an exudation of lymph and serum principally be-

neath and on the free surface of the membrane covering the valves and chordæ tendinæ. As the disease progresses, large or small papillary nodules, vegetations, are formed on the endocardium—endocarditis verrucosa; or ulcerations may occur as a result of destruction of the superficially necrosed tissue—endocarditis ulcerosa. The latter condition is usually found in the malignant, usually septic, form of endocarditis. During the course of endocarditis the pericardium and myocardium become involved and many organs of the body, e. g., the kidneys, spleen, brain, etc., may become implicated through emboli composed of masses of fibrin or necrosed tissue which become detached by the circulating blood, principally from the irregular valvular deposits. In septic cases these emboli give rise to abscesses.
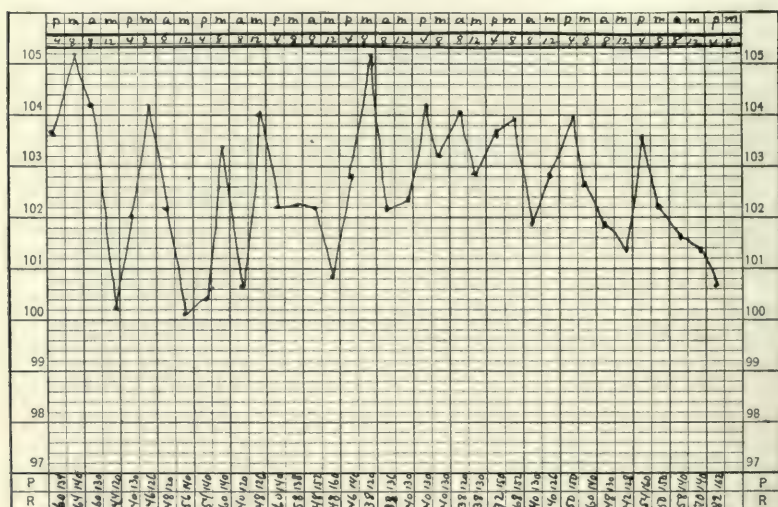
Fig. 154.—Fever curve of malignant endocarditis in a child three years old.

It is well to remember, however, that moderately severe cases of endocarditis may go on to complete recovery and leave no trace of the original inflammation on the endocardium; furthermore, that slight valvular vegetations are not infrequently found postmortem without any apparent clinical signs of heart disease during life. This latter observation can readily be explained by the fact that mild endocarditis is not rarely masked by the course of another disease and, unless presenting marked disturbance of the circulation, is very apt to be overlooked. More often, of course, endocarditis sets in with severe unmistakable symptoms. The patient vomits, suffers from chills, more or less high fever (102° to 105° F.), precordial distress, short

cough, dyspnea, and accelerated, sometimes irregular, pulse. These symptoms, however, are not sufficiently characteristic of endocarditis and may still leave the nature of the disease obscure until the subsequent appearance of local signs, especially of a systolic heart murmur, audible chiefly at the apex (the mitral valve being most frequently involved owing to its great vascularity) or also over the whole cardiac region. Sometimes endocarditis follows an apparently mild attack of tonsillitis with possibly a moderate joint involvement, with or without signs of chorea. As will be seen later (see "Endocarditis Chronica," p. 536), murmurs may subsequently develop at the various orifices of the heart, and, at a later stage of the disease, additional physical signs (hypertrophy or dilatation) may be obtained by percussion.

Occasionally (in children less frequently than in adults) acute endocarditis pursues a very septic and often violent course—*endocarditis maligna (ulcerosa)*. It may be preceded by pneumonia, exanthematous diseases, septic processes in some other parts of the body, *e. g.*, osteomyelitis, etc., or occur without any apparent cause and exhibit a symptom complex resembling either a low typhoid state or cardiac insufficiency with acute dilatation (cyanosis) and loud murmurs at the various orifices. The duration of malignant endocarditis varies. Ordinarily it runs a protracted course with irregular temperature, chills, rigors and sweats. Sooner or later emboli develop in different organs of the body and the capillaries of the skin, the superadded symptoms varying, of course, with the organ affected. Thus, if the brain is involved, we find palsies with disorder of consciousness; if the spleen, enlargement of this organ and tenderness; if the kidneys, albuminuria, hematuria and anasarca; if the skin, petechiæ and a pustular eruption. It is not rarely complicated also by purulent pericarditis. When malignant endocarditis runs so very violent a course it, as a rule, terminates fatally within a few days. On the other hand, simple, benign endocarditis in children is usually not dangerous to life. If free from complications, the symptoms begin to subside after about a week or ten days, eventually leading to recovery in about four weeks. In quite a number of cases, however, it is followed by permanent valvular disease, with or without cardiac hypertrophy. (See "Endocarditis Chronica," p. 536.) Death is usually due to cardiac paralysis.

Benign endocarditis may be mistaken for dry pericarditis, especially if the former is associated with articular rheumatism. The following table contrasts the most important distinguishing features. Both diseases, however, may coexist.

Simple Endocarditis	Dry Pericarditis
Blowing or musical sound.	"To and fro" friction or creaking sound.
Sound is associated with systole or diastole.	Not necessarily. May be heard at any period of cycle.
Sound is distant.	Near to the ear.
Sound is uninfluenced by pressure with the stethoscope.	Increased.
Sound is conducted upward, to the axilla, and to the back.	Not so.
Sound usually loudest at apex.	Anywhere over precordium.

The diagnosis of ulcerative endocarditis is very difficult, especially in the incipient stage, before the appearance of a heart murmur. Whenever several orifices are the seat of the murmur, paroxysms of cyanosis, prostration, dyspnea, and irregular temperature predominate and cardiac dulness is increased, the diagnosis of malignant endocarditis is justified. The elimination of typhoid, irregular malarial fever, military tuberculosis and pyemia, the four affections with which malignant endocarditis is most apt to be confounded, will greatly facilitate the diagnosis.

Treatment.—The treatment of endocarditis is essentially the same as in pericarditis—purely symptomatic. Absolute rest in bed, in the recumbent posture, and a light ice bag to the precordium. Antirheumatic remedies, in conjunction with small doses of codeine and digitalis to strengthen the heart. Light (meat free) easily assimilable diet. Cool sponging for high temperature. In malignant endocarditis some benefit may be derived from polyvalent antistreptococcus serum and transfusion. As recurrent endocarditis is not rarely due to persistent infection of the nasopharynx, it is always in order to use a nose and throat wash a few times daily, irrespective of the cause. (See "Pericarditis," p. 533.)

Endocarditis Chronica

(Valvular Heart Disease)

Chronic endocarditis is most frequently a sequel of acute inflammation of the endocardium especially of the valves, and pathologically consists of proliferation and thickening of the valvular connective tissue with a great tendency to contractions and adhesions and very rarely to calcification. The chronic inflammatory process is usually limited to the left side of the heart except in cases developing during fetal life, when the reverse is the case.

Coincident with the inflammatory process in the endocardium, the

cardiac musculature undergoes gradual enlargement. This hypertrophy, unless assuming exceptionally large dimensions (*e. g.*, cor bovium), is strictly speaking not a disease *per se*, but on the contrary, an effort on the part of nature to overcome or undo the evil effects of the disease. As the disease advances and the heart muscles lose their
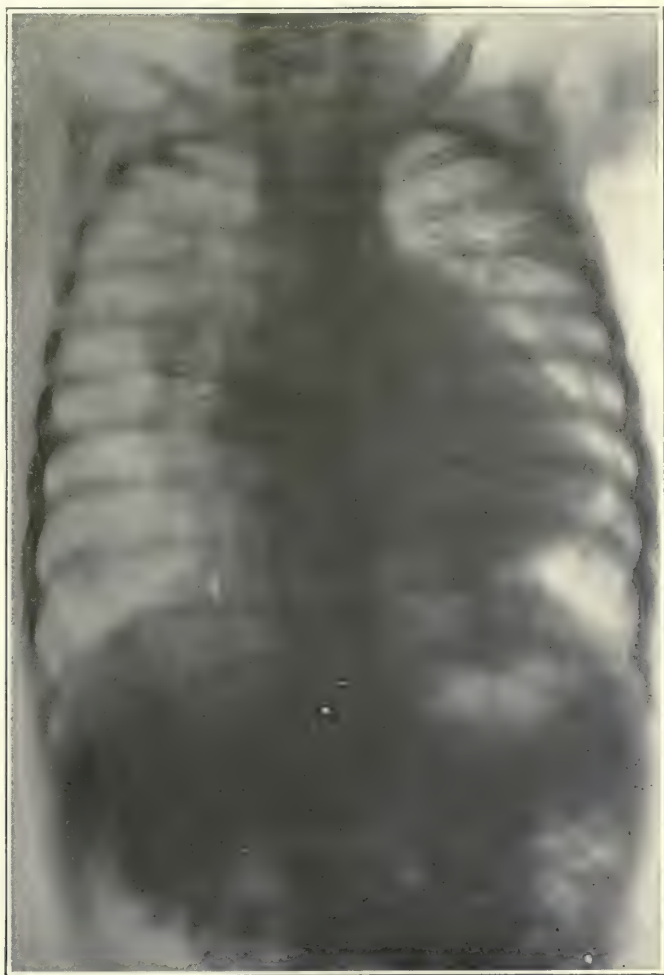
Fig. 155.—Intense dilatation of the heart in a two-months-old infant suffering from congenital heart disease which was greatly aggravated by an attack of whooping cough.

power, get exhausted, the hypertrophy is replaced by dilatation, indicating that compensation has "ruptured," and that the disease is beyond control.

Until failure of compensation has occurred, children may for years remain apparently free from any marked disturbances of health, except perhaps, an indistinct first sound at the apex, an unduly bounding pulse, throbbing of the blood vessels in the neck, rapid fatigue, palpitation of the heart on exertion, progressive anemia and malnutrition notwithstanding good appetite and digestion. Indeed, it is often for disturbance of the stomach that the patients are brought to the physician. Shortness of breath, which increases on exertion, usually forms an early manifestation of failing compensation. It is the result of stasis in the pulmonary circulation with consecutive impairment of aëration. This sooner or later leads to passive congestion of the pulmonary alveoli giving rise to bronchitis with an irritable cough, and as the heart failure increases, to paroxysmal attacks of dyspnea or orthopnea especially at night ("cardiac asthma") pulmonary edema, cyanosis, and occasionally to hemorrhagic infarcts in the lung with consecutive hemoptysis.

Simultaneously with the aforementioned manifestations pathologic changes go on also in other internal organs—the liver, spleen and kidneys. The liver and spleen are enlarged, and by pressure upon neighboring thoracic and abdominal organs, increase the dyspnea. As a sequel of the passive congestion of the liver and stasis in the blood vessels of the stomach and intestines, numerous gastrointestinal disturbances—e. g., anorexia, vomiting, constipation—develop, which add misery to the patient's painful existence.

The changes in the kidneys are manifested by diminution in the quantity of urine, often albuminuria (slight), hyaline and cylindrical casts, and occasionally white and red blood corpuscles—signs of passive congestion.

With increasing venous stasis there is coincident transudation of the fluid of the blood from the capillaries into the meshes of the tissues, leading to edema. At first the dropsy is limited to the ankles and eyelids, but as the disturbance of circulation advances it grows worse and involves the entire integument and the internal cavities, especially the abdominal and pleural cavities. Notwithstanding the extreme gravity of the condition, the end is not always as near as would be expected. The inherent power of the infantile heart is still capable of temporary reparation. The arrhythmia, dyspnea, and dropsy may disappear; the appetite and nutrition may improve; the tottering patient may again be up and around, in fact, may appear at his best.

Exacerbations and improvements of the disease may recur several times. The improvement, as a rule, is but short lived. Very soon the symptoms return, and generally with greater severity. Finally, after

a more or less prolonged period of illness the patient succumbs to heart failure. Occasionally, death occurs suddenly after severe exertion. Quite a number of children are carried away by intercurrent infectious diseases, pericarditis or recurrent acute endocarditis. The physician should therefore always be very guarded in the prognosis. The relative gravity of valvular lesions is as follows: Tricuspid regurgitation; mitral regurgitation; mitral stenosis; aortic regurgitation; pulmonic stenosis; aortic stenosis.

Differential Diagnosis*

As the physical signs of valvular heart disease in children differ but little from those in adults, we will briefly review only the most essential differential points of diagnosis.

Mitral Regurgitation.—Insufficiency of the mitral valve is characterized by a systolic blowing murmur which is loudest at the apex and transmitted to the axilla and near the lower angle of the left scapula. Accentuation of the second pulmonic sound. Hypertrophy of the left ventricle, and later left auricle, and sequential hypertrophy of the right ventricle. The pulse may be normal or accelerated, and with disturbed compensation—which may not occur for many years—irregular and unequal.

Mitral Obstruction.—It is frequently associated with insufficiency. The murmur is usually presystolic or also diastolic, is best heard at the apex, and may be conveyed to the fourth interspace, but never to the angle of the scapula. The pulmonic second sound is accentuated and sometimes double. It frequently leads to hypertrophy of the left auricle and right ventricle.

Aortic Regurgitation.—Aortic insufficiency is rare in children. It is accompanied by hypertrophy of the left ventricle, and often pulsation of the arteries of the neck. The murmur is diastolic, loudest at the insertion of the right second costal cartilage and over the upper portion of the sternum. At first the murmur is quite noisy, but with ensuing disturbance of compensation it loses its intensity. It is usually combined with aortic stenosis, becoming the gravest form of valvular disease of childhood. It sometimes causes sudden death, and but few children survive the age of puberty. Aortic regurgitation may often be recognized by the peculiarly collapsing pulse, the water-hammer or Corrigan's pulse.

Aortic Obstruction.—This affection is usually observed in older children in connection with aortic insufficiency. The murmur is harsh, systolic, heard loudest over the orifice, transmitted to the right,

---

*See Fig. 21.

and sometimes over the whole length of sternum, and the arteries of the neck. Hypertrophy of the left ventricle.

Tricuspid Regurgitation.—Except as a congenital defect, it most frequently occurs secondarily to affections of the left heart. Auscultation reveals a systolic blowing murmur, heard loudest over the lower part of the sternum (xiphoid) and at the juncture of the fourth costal cartilage. Second sound is weak. Jugular pulsation. Hypertrophy and dilatation of the right heart. In severe cases cyanosis, and pulsation of the liver.

Tricuspid Obstruction.—This condition is extremely rare as an acquired heart affection, hence calls for no detailed discussion. No particular change in size of the heart is known. (See ''Congenital Heart Disease,'' p. 528.)

Pulmonic Regurgitation.—Insufficiency of the pulmonic valve is chiefly congenital, rarely acquired. The murmur is diastolic and limited to the site of the valve—at the juncture of the left second costal cartilage and the sternum. Unlike that of aortic insufficiency it is not transmitted to the arteries of the neck. Hypertrophy of the right heart.

Pulmonic Obstruction.—Principally a congenital malady. The murmur is basic, systolic, heard loudest at the left second costosternal junction. It is associated with hypertrophy of the right ventricle, and sometimes with cyanosis. (See ''Congenital Heart Disease.'')

Treatment.—The management of chronic valvular heart disease in children is the same as in adults. It differs with the stages of the disease—when compensation is intact, and when it ''ruptures.''

Stage of Compensation

The well-being and longevity of the patient stand in direct ratio to the capacity of the heart to compensate its insufficiency by secondary hypertrophy of the musculature of one or more of its chambers. Hence, the aim in the treatment of chronic valvular heart disease should be directed chiefly to the maintenance of compensatory hypertrophy. Bearing in mind the facts that with increasing circulatory disturbance there is on the part of the heart a spontaneous muscular development to overcome its difficulties so long as its supply of nourishment is sufficient and its hypertrophic process is not interfered with by unequal demands upon its reserve force, as is apt to occur, *e. g.*, in overexertion, in intercurrent diseases and the like, we can readily formulate a plan of treatment which will at least for a time, amply meet with the aforementioned indication. Parents should be given to understand that the treatment of compensating heart disease is principally prophylactic and hygienic and that its success is

commensurate with the degree of cooperation on the part of the patient as well as those guiding his destiny, when the heart is at its best. Convalescence from acute or recurrent heart disease calls for very careful attention. Too early attempts at walking or standing are apt to prove disastrous, not rarely leading to sudden dilatation of the heart, perhaps with fatal issue. Beginning with gradual raisings of the patient's head and shoulders, and watching its effect upon the patient's heart action—its strength and rhythm—we may gradually allow greater liberties, provided slight exertion is unattended by detrimental influences. In severe cases of valvular heart disease it is usually not safe to permit the patient to be out and around in less than three months. A sojourn in a quiet inland resort is very helpful to recovery.

A heart with crippled valves demands an adequate supply of healthy blood in the coronary arteries. This is best secured by suitable nutrition and a rational mode of living. The diet must be appropriate to the age of the patient, at all ages milk, cereals and vegetables forming the principle food ingredients; eggs, fish, light meats and fruit may be added off and on. Liquors and stimulants of all kinds should be avoided, administering instead nutrient tonics such as malt and cod liver oil, with or without small quantities of iron and arsenic, etc.

Special attention should be paid to the action of the bowels, kidneys, and skin. Daily cool sponging followed by gentle massage is very invigorating. Warm clothing is essential, but unnecessary coddling of the patient should be interdicted. Weather permitting, the child should be kept outdoors from nine in the morning until five (later in the summer) in the afternoon, allowing him to participate in all such amusements as will not call for undue exertion. Racing, jumping, football, and baseball playing and swimming should be forbidden. Light athletic exercise is useful, if it gives rise to no undue fatigue, or disturbance of compensation (see p. 542). Passive exercise in the form of massage is highly to be recommended. The question of how much brain work a patient with poorly compensating heart disease is to be permitted to do, cannot be decided offhand, to apply to all cases. Its effect upon the general health of the patient must be watched, and changes in the curriculum promptly made if headache, insomnia, anemia, debility, excessive nervous irritability, and the like, make their appearance.

It is of very vital importance to obviate intercurrent diseases, especially infectious diseases, such as scarlatina, articular rheumatism, etc., which are apt to reinfect the endocardium and aggravate the pa-

tient's condition. If such diseases prevail it is imperative, whenever practicable, to isolate the child, or remove him to a place where he will be least exposed to infection. For fear of contracting contagious diseases patients in good financial circumstances should be kept from visiting public or private schools and preferably be instructed at home.

Particular attention should be paid to incipient symptoms of tonsillitis, ''growing pains,'' etc.,—forerunners of rheumatism. In these conditions the salicylates should be resorted to early, to prevent graver rheumatic manifestations. Hypertrophied tonsils and decayed teeth should receive special care.

With every appearance of indisposition the patient should be put to bed, and kept there until every vestige of the malady has abated.

In intercurrent febrile diseases the heart demands very careful watching, and in the presence of any disturbance, immediate treatment.

Formal Gymnastics—Cardiac Cases*

These educational and hygienic exercises, as the terms are applied in public school systems, are examples of the type of exercise to be used in alternation, so as to change the groups of muscles employed and the vigor. The number of times and the vigor with which the exercise is done, will increase the effort required of the heart. The teacher must use her judgment with regard to the effect on individual cases. The children must be taught to discontinue exercising at any moment subjective symptoms become marked.

Drill I—Duration 15 minutes.
  1. Hands on shoulders—Place!
      (1) Stretching left arm upward, right arm downward; hands on shoulders; stretching right arm upward, left arm downward—Begin!
  2.   (1) Point step forward left, raising arms forward—One!
      (2) Point step sideways left, arms sideways (palms up)—Two!
      (3) Point step backward left, arms upward—Three!
      (4) Replacing foot, arms forward, downward—Four!
      Name of exercise—Point step forward left, arms forward; point step sideways left, arms sideways; point step backward left, arms upward.
  3. Hands on hips—Place!
      (1) Deep knee bending—One!
      (2) Stretching knees—Two!
      This exercise should be taught to response commands and after it is thoroughly learned should be done in rhythm. Then the exercise is— Hands on hips—Place!
      (1) Deep knee bending—Begin!

---

*The author is indebted to Dr. Robert H. Halsey for the following outline of the graduate exercises used in the cardiac clinics for children.

4. (1) Raising arms sideways—One!
    (2) Bending trunk sideways left—Two!
    (3) Trunk erect—Three!
    (4) Position—Four!

    <center>Same right. Alternate.</center>

    Name—Raising arms sideways; bending trunk sideways.
5. Arms sideward. Trunk to right—bend. (Repeat to left.) Trunk raise.
6. (1) Placing left foot sideways, raising arms sideways—One!
    (2) Bending trunk forward, bending left knee, touching left hand to left toe,
        right arm upward—Two!
    (3) Same as (1)—Three!
    (4) Position—Four!

    <center>Same right. Alternate.</center>

    Name—Placing left foot sideways, arms sideways; bending trunk forward,
        bending left knee, touching left hand to left toe right arm upward.
7. (1) Raising heels and arms sideways—Begin!
8. Hands at side of shoulders—Place!
    (1) Stretching arms upward; hands at sides of shoulders; stretching arms
        sideways (palms up)—Begin!

Drill II—Duration 10 minutes additional, making 25 minutes in all.
1. (1) Placing left foot sideways, bending arms at shoulder level—One!
    (2) Position—Two!

    <center>Same right. Alternate.</center>

2. Hands on hips—Place!
    (1) Bending head backward (slowly)—One!
    (2) Raising head (slowly)—Two!

    <center>Repeat.</center>

3. (1) Deep knee bending, raising arms sideways—Begin!
4. (1) Placing hands behind neck—One!
    (2) Bending trunk sideways—Two!
    (3) Trunk erect—Three!
    (4) Position—Four!

    <center>Same right. Alternate.</center>

5. Hands on hips—Place!
    (1) Raising left leg forward—One!
    (2) Lowering left leg to position (slowly)—Two!

    <center>Same right. Alternate.</center>

6. Hands on hips—Place!
    (1) Springing feet sideways—Begin!
7. Hands on hips—Place!
    (1) Raising left leg sideways (slowly)—One!
    (2) Position—Two!

    <center>Same right. Alternate.</center>

8. Hands on shoulders—Place!
    (1) Raising heels, stretching arms upward—Begin!

Drill III—Duration 5 minutes additional, making 30 minutes in all.

1.   (1) Clapping hands over head—One!
       (2) Raising left knee, clapping hands under left knee—Two!
       (3) Replacing left foot, clapping hands over head—Three!
       (4) Hands on hips—Four!
<div align="center">Same right. Alternate.</div>

2. Hands on hips—Place!
       (1) Hopping twice on each foot, starting left—Begin!

3. Prone fall position. Walk forward. Forward jump. Stand.

4. Hands on hips—Place!
       (1) Raising knees upward, alternately, starting left in quick rhythm—Begin!

5.   (1) Raising heels, arms sideways—Begin.

Stage of Failing Compensation

Varying with the inherent strength of the patient, the severity of the lesion and the precautionary measures employed, compensation may be maintained for a shorter or longer time—weeks, months, or years. However, it is only a question of time when compensation ruptures. As previously mentioned, the breakdown may be only temporary, (readily yielding to a few weeks of rest, careful feeding and possibly requiring also a few doses of digitalis) and recur on several occasions. But sooner or later the heart muscle gives way, the pulse becomes feeble and irregular, the breathing deep and difficult, the urine diminished in quantity and the general health of the patient greatly impaired. Here rest in bed is indispensable, but this alone is not sufficient to restore compensation. We have to resort to cardiac stimulants to strengthen the heart muscle and to regulate its beat, and also to vasodilators, to allow the blood to flow in the arteries without resistance, with each ventricular contraction. Various drugs are being recommended for this purpose, but none meets the indications with the same degree of certainty as digitalis, and the iodides. In incipient failure of compensation we usually begin, for every two years of the child's age, with $\frac{1}{2}$ grain of the sodium iodide and $\frac{1}{4}$ dram of the infusion of digitalis, or one drop of the tincture, to be repeated every six hours; and as the disease advances we increase the doses proportionately up to 1 grain of the iodide and 1 dram of the infusion of digitalis or 2 drops of the tincture. The cumulative action of the digitalis should be borne in mind and its administration discontinued if untoward symptoms arise. In this event, or where digitalis is not well tolerated by the stomach, we may substitute strophanthus, diuretin, caffeine sodium benzoate or spartein sulphate instead. The latter two remedies have the advantage that they may be safely given hyperdermically if irritability of the stomach precludes their administration by mouth. In the early attacks of failure of compensation the benefits obtained from the simple mode of treatment just outlined

are often entirely beyond expectation. Sometimes within but a very few days the urine greatly increases in quantity, the edema disappears, the dyspnea ceases, the distressing cough abates; in short, restoration of compensation is apparently complete. In the later stages of compensatory failure, however, the treatment by means of rest, good food, the iodides and digitalis fails to assert its magic influence. We have to resort to symptomatic medication, especially with the view of relieving suffering. In this respect the treatment is the same as that employed in adults, morphine with or without atropine, by mouth or hypodermically, being the most potent remedy at our command.

R Strychninæ Sulph. gr. ⅛ | 0.008
Natrii Iodidi gr. xvj | 1.000
Inf. Digitalis fol. ℥ j | 30.000
Syr. Altheæ q. s. ad ℥ ij | 60.000
M.
    S.—One teaspoonful t. i. d., for a child four years old. (Alterative heart tonic.)

R Syr. Ferri Iodidi ℥ iij | 12.0
Syr. Aurantii q. s. ad ℥ ij | 60.0
M.
    S.—One teaspoonful every four hours, for a child four years old. (Between ''heart attacks.'')

R Liq. Ferri et Ammonii Acetatis
Inf. Digitalis fol. āā ℥ j | 30.0
M.
    S.—One teaspoonful every four hours, for a child four years old. (When dropsy is present.)

R Tr. Digitalis
Tr. Strophanthi āā ℥ ij | 8.0
M.
    S.—Five to ten drops every four hours, for a child four years old. (In marked heart dilatation with irregularity.)

R Strychninæ Sulph. gr. ⅕ | 0.012
Caffeinæ Natrii Benzoatis gr. xij | 0.800
Aq. Destil. ℥ ij | 8.000
M.
    S.—Ten drops hypodermically, p. r. n., for a child four years old. (Quick stimulant.)

A light diet is essential. Skimmed milk (Karell's diet for heart disease), 3 to 6 ounces every four hours, is often very beneficial. This diet may be strengthened by the addition of cereals and lactose.

DISEASES OF THE BLOOD AND DUCTLESS GLANDS

DISEASES OF THE BLOOD

Affections of the blood are of very common occurrence in children, especially in infancy and in those approaching puberty. At these periods of life, owing to the rapid bodily development, the blood-forming organs are taxed to their greatest capacity, and, hence, are very apt to suffer on slight provocation. The anemias of children are usually secondary in nature, only exceptionally primary. With the present inadequate state of our knowledge, however, no sharp line of demarcation can as yet be drawn between the various types of blood disease. Only too often do we find the clinical and histologic aspects of simple secondary anemia merging into that of splenic anemia, and that of the latter disease into the one of leukemia. The same is true of lymphatic leukemia, chloroma, and lymphosarcoma. For the reasons just stated, therefore, no attempt will here be made to offer an iron-clad classification of the diseases in question.

In studying blood disease it is well to bear in mind that the constituents of the normal blood vary within more or less wide limits, and that slight ailments are prone to produce marked disproportion between the number of red and white blood corpuscles.

At birth the number of red cells is about 6,000,000, and of white cells, between 20,000 to 30,000 per cubic millimeter. The hemoglobin is very high, about 110 per cent and the specific gravity 1,066. After the second week the red cells fall to 5,000,000, and the white cells to about 15,000, the hemoglobin to 100 per cent, and the specific gravity to 1,050. The red cells are fewer in number in the female than in the male. The *percentage* of the different leucocytes in infants presents the following variations: Polymorphonuclear neutrophiles, 28 to 50; polymorphonuclear eosinophiles, ½ to 10; lymphocytes, 50 to 70, and large mononuclears, 6 to 14. The adult proportion is usually reached by the time the child is eight years old. Then the number of polymorphonuclears rises to 65 or 75 per cent and that of the lymphocytes falls to 20 or 30, and of the mononuclears, to 1 to 4. Normally, coagulation of the blood usually occurs within from two to five minutes.

DIFFERENTIAL BLOOD COUNTS IN NORMAL CHILDREN

(After Schloss)

AGE		POLY-MORPHO-NUCLEARS	LYMPHO-CYTES	LARGE MONO-NUCLEARS	EOSINO-PHILES	BASO-PHILES
months 6-12	Maximum	35.9	58.5	12.2	4.5	0.8
	Minimum	24.6	50.5	7.3	0.0	0.1
	Average	30.4	55.9	9.6	2.6	0.4
1-2 yrs.	Maximum	39.7	58.8	11.7	5.0	0.5
	Minimum	27.5	45.3	6.7	1.6	0.0
	Average	36.3	51.2	8.5	3.2	0.2
2-3 yrs.	Maximum	44.3	55.0	11.3	0.5	1.2
	Minimum	33.2	43.5	5.0	3.1	0.0
	Average	38.7	49.9	8.2		0.4
					6.0	
3-4 yrs.	Maximum	54.1	47.6	16.2	4.2	0.9
	Minimum	36.2	32.2	6.0	1.5	0.0
	Average	44.7	39.1	11.2	2.8	0.5
4-5 yrs	Maximum	51.7	49.5	6.7	4.0	0.6
	Minimum	42.2	38.4	3.4	1.6	0.3
	Average	48.5	42.1	6.0	2.6	0.3
5-6 yrs.	Maximum	61.8	36.7	16.0	4.7	1.0
	Minimum	52.6	21.2	6.5	0.7	0.3
	Average	56.5	29.9	10.0	2.5	0.6
6-7 yrs.	Maximum	61.3	34.1	15.7	4.7	0.6
	Minimum	52.3	24.5	8.1	0.1	0.0
	Average	56.0	30.4	10.8	2.2	0.2
7-8 yrs.	Maximum	72.0	39.1	15.2	3.5	0.2
	Minimum	45.2	21.1	6.7	0.0	0.0
	Average	54.4	32.5	11.6	6.1	0.6

Anemia Simplex, Chlorosis

(GREEN SICKNESS)

Both of these conditions present identical pathologic changes in the blood—reduction in the number of red cells, decrease of hemoglobin, without marked changes in the cells themselves—but differ somewhat in the etiology and course. Thus, while chlorosis is ordinarily encountered in girls at puberty, and almost invariably ends in recovery without any grave alterations in the general health, anemia is a disease of younger children, and if occurring in infants very frequently forms the forerunner of that type of blood disease which is generally described as pseudoleukemia infantum (q. v.). Anemia in the newborn may be congenital (disease of the mother, especially syphilis, tuberculosis, and malaria) or acquired (hemorrhage; sepsis).

Anemia as well as chlorosis is manifested by pallor of the face (waxy or green complexion) and mucous membranes, headache, dys-

peptic symptoms, undue fatigue after slight exertion, attacks of palpitation of the heart and of dyspnea, general debility and excessive irritability of the nerve system. In young infants the ears usually show a peculiar waxy transparency. Auscultation often reveals hemic murmurs along the large veins of the neck and at the base of the heart, which differ from organic murmurs by their inconstancy and frequent change in their intensity and location.

In addition to the aforementioned manifestations, chlorosis in mature girls is very prone to give rise to amenorrhea, dysmenorrhea, and less frequently to menorrhagia with consequent aggravation of the original condition; severe chlorosis is apt to be complicated by venous thrombosis, especially in the lower extremities and the brain sinuses, and occasionally by secondary gangrene and embolism. Of course such occurrences are very exceptional. The very great majority of cases of chlorosis, as already stated, improve rapidly and fully, although relapses are not uncommon.

The **management** of anemia and chlorosis to a great extent varies with the numerous etiologic factors. The general health should be improved by suitable nutritious diet, plenty of outdoor air, in older children cold shower baths with gentle massage, ample sleep, and avoidance of undue excitement and physical and mental overexertion. Dyspepsia, habitual constipation, diarrhea, loss of blood (epistaxis, etc.), hereditary syphilis, malaria, tuberculosis, heart and kidney affections, and all other diseases as are apt to undermine the system should receive prompt and continuous attention. Where circulatory disturbances are very pronounced, rest in bed is indispensable. Medicinally, iron and arsenic are the remedies of choice. The following combination acts splendidly :

℞	Liquoris Arsenici Chloridi	ℨ i	4.00
	Tr. Ferri Chloridi	ℨ iii	12.00
	Syr. Aurantii	q. s. ad ℥ iii	90.00
	M.		

    S.—One teaspoonful every three hours, for a child six years old.

In older children to avoid destruction of the teeth, the iron and arsenic, without syrup, may be prescribed with instructions to be taken in capsule form, each dose being prepared before taking it in accordance with the directions given on page 104.

Digestives and tonics (cod liver oil) will be found to act as useful adjuvants. Change of air, preferably to mountainous regions.

Pseudoleukemia Infantum, Splenica

(VON JAKSCH'S OR SPLENIC ANEMIA)

This condition was first described by von Jaksch in 1889 as a clinical entity. It is observed in infants from six to twenty-four months of age, corresponding with the age when rachitis and gastrointestinal affections are most prevalent. Hence the reason why some authors look upon it as a secondary rather than a primary anemia.

The chief alterations in the blood are reduction of red cells and hemoglobin (sometimes as low as 20 per cent), the presence of many

Fig. 156.—Splenomegaly in association with von Jaksch anemia.

nucleated red corpuscles, and a considerable increase in the number of leucocytes, mostly of the mononuclear type. This blood picture essentially corresponds to that of ordinary secondary anemia. In pseudoleukemia infantum, however, there is an enormous enlargement of the spleen and usually also slight enlargement of the liver and lymphatic glands.

The general symptoms differ but little from those observed in severe anemia. The same applies to the treatment. The syrup of the iodide of iron with the syrup of the hypophosphites and red bone marrow seem to exert a specific action in the majority of cases.

Pseudoleukemia Lymphatica

(HODGKIN'S DISEASE, ADENIE, LYMPHADENOMA)

In contrast to splenic anemia this disease is not peculiar to infancy and is characterized by multiple hyperplasia of the lymph glands with progressive anemia. The cervical glands are most commonly and severely attacked, but the lymphoid tissue of the entire body is more or less involved. It closely resembles tuberculous adenitis, except that it is much more uncommon than tuberculosis and that in the latter condition the glands show a greater tendency to caseation and suppuration. In doubtful cases the tuberculin test may prove decisive in the diagnosis.

The changes in the blood and the clinical manifestations are identical with those observed in severe anemia. Occasionally, there are local pressure symptoms, such as pain, edema, cough and dyspnea.

Under suitable treatment (see "Anemia," p. 548) recovery or at least arrest of the disease is possible. Of late arsenic in the form of salvarsan has been employed with considerable success. X-ray therapy also is worth trying and some clinicians advise surgical intervention. Intractable cases often terminate in leukemia.

Leukemia

(LEUCOCYTHEMIA)

As the term indicates, leukemia is characterized principally by an abnormal increase in the number of leucocytes (sometimes reaching as high as a million), and by the presence of unusual types of these cells, i. e., "Markzellen" (myelocytes), "Mastzellen" (nutritive cells), and giant basophiles. From a pathologic point of view it is customary to distinguish two forms of leukemia: (1) Lymphatic leukemia, in which the lymphatic glands are chiefly involved (hyperplasia); and (2) splenomedullary or myelogenic form, in which the spleen (greatly increased in size) and the bone marrow (hyperplasia) are the principal seats of the lesion. Mixed forms also are encountered. The principal difference between the two forms of leukemia are the preponderance of lymphocytes in lymphatic, and myelocytes in splenic leukemia. The red cells and hemoglobin are reduced in both varieties.

The clinical manifestations are essentially identical with those of pernicious anemia (q. v.), plus enlargement of the lymphatic glands, (of the neck, axilla and inguinal regions), the spleen and liver. There is a marked tendency to hemorrhage in the skin and mucous membranes; progressive anemia; recurrent fever; edema. The disease may run a very acute course (acute leukemia), and end fatally within a week or two, or proceed a slower course (chronic leukemia), and lead to a fatal issue after a few months.

As the nature of leukemia is entirely obscure, little else can be done but treat it symptomatically. (See Anemia and Pseudoleukemia.)

Pernicious Anemia

This form of anemia is characterized by great diminution in the number of red cells (2,000,000 to 1,000,000 per c. mm.); reduction in the total quantity of hemoglobin with a comparative increase of the hemoglobin in the red cells; increase in the size of the red cells with predominance of megaloblasts; loss of cohesive quality of the red cells (their failure to form rouleaux), and, finally, absence of distinct changes (or slight reduction) in the number of the leucocytes.

This blood affection is very rarely met with in children. As in adults, it may occur secondarily to protracted simple anemia or in consequence of abstraction of blood by intestinal parasites, e. g., bothriocephalus latus; uncinaria (q. v.).

In the beginning the symptoms resemble those of severe simple anemia (q. v.), but at a later stage of the disease the condition is greatly aggravated by supervening hemorrhages from the mucous membranes, cutaneous ecchymoses, and general dropsy. In such cases death invariably occurs within a few months.

Pernicious anemia occasionally gives rise to lesions in the spinal cord with corresponding symptoms (paralysis of the extremities, etc.).

Postmortem examination usually reveals fatty degeneration of the internal organs.

The Treatment.—The treatment is the same as in severe anemia. (See p. 548.) In addition, removal of the intestinal parasites, if present, and transfusion.

A. D. Espine (Rev. Méd. de la Suise Romande, August, 1918) reports the recovery of two young infants treated by a special serum. The serum was obtained by venesection of animals at the height of the regeneration of blood following a previous extensive withdrawal of blood. This "hematopoietic serum" was injected subcutaneously daily in doses of from 5 c.c. to 10 c.c. The improvement was gradual.

Hemorrhea Congenita

(Hemophilia)

Hemophilia is an inherited, congenital tendency to posttraumatic or spontaneous, profuse, often uncontrollable, hemorrhage. It affects the male much more frequently than the female, though the disease is transmitted through the female. The disease becomes less marked with advancing age.

According to Haliburton, the process of blood coagulation is as follows:

Blood platelets and leuco-cytes give thrombogen	The blood cells and tissues of the body give thrombokinase
Blood plasma furnishes a protein fibrinogen	In the presence of calcium salts thrombokinase activates thrombogen into an enzyme

Thrombin acts on fibrinogen to give

Fibrin
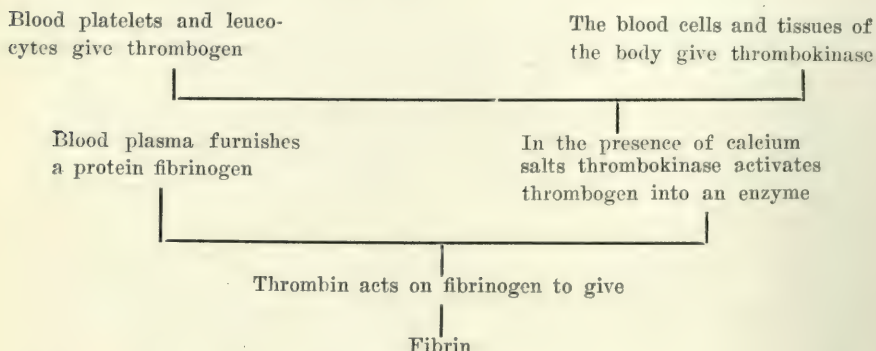
Minot and Lee maintain that the active coagulating principle of the tissue juice is derived in part, if not wholly, from the blood platelets and that in hemophilia there is an hereditary defect in the platelets, though normal in number. In addition to this there is also a congenital permeability and friability of the blood vessels.

While, as previously alluded to, the hemorrhage may start spontaneously, in the great majority of cases it follows some trivial injury. A scratch or the prick of a pin or slight abrasion of the body surface, vaccination, snipping of the frenum linguæ, circumcision, extraction of a tooth, opening of abscesses, etc., are followed by severe, often by uncontrollable, hemorrhage. Any undue exertion of a muscle or a group of muscles (e. g., jumping off a chair, sudden twisting of an arm), a bump or a blow, etc., often gives rise to a profuse extravasation of blood into the skin or joints. Forcible blowing of the nose may be followed by an exsanguinating nosebleed, and in a case under observation sneezing produced an enormous hemorrhage from the nose and ear (rupture of the drum!) which nearly ended fatally. In girls hemorrhages may occur from the vagina (often mistaken for menstruatio precox) long before the age of puberty; and with establishment of menstrual function, the bleeding may be so profuse as to

leave the patient monthly in a state of collapse. Hematemesis, hemorrhage from the bowels and hematuria are less common, and bleeding into the serous cavities (peritoneal, pleural and pericardial) and the brain are still less frequent. Hemophilia in the newborn may be manifested during or immediately after birth by severe hemorrhages occurring from abrasions and contusions sustained during delivery, or after cutting the umbilical cord. These hemorrhages are not to be mistaken for hemorrhage in the newborn complicating sepsis (see p. 229), or the so-called transitory hemophilia which is manifested by idiopathic umbilical hemorrhage (see p. 222), or fearful, sometimes fatal bleeding following ritual circumcision. In this form of hemophilia the tendency to hemorrhage is greatest between the seventh and fourteenth days of life, gradually lessening in intensity until the infant reaches the age of two or three months, when it disappears entirely. The differential points of diagnosis between hemorrhea congenita and hemorrhea acquisita will be spoken of in the discussion of the latter affection.

Treatment.—Little of a permanent cure can be expected from treatment, except in mild forms of hemophilia (''partial bleeders''). In these cases gelatine as a food, and calcium chloride, in from 2 grain to 5 grain doses, twice daily, to be continued for months or years, will prove of some benefit. Thyroid gland substance, in small doses, continued for weeks at a time, is deserving of trial. For the immediate arrest of the hemorrhage we must resort to transfusion or injection of whole blood (2 or 3 ounces two or three times a day) into the gluteal region. In slight local hemorrhage good results are often obtained from the topical application of thrombokinase and thromboplastin.

We should guard against injuries and operative interference (gelatin feeding before operation is helpful) of all kinds.

Bleeders, especially females, should not marry.

Hemorrhea Acquisita

(Purpura Simplex, Purpura Hemorrhagica s. Morbus Maculosus, Purpura Fulminans)

Purpura is an acquired affection of the blood or its vessels characterized by hemorrhage into the skin, mucous membranes and other tissues, and by more or less marked constitutional disturbance.

The etiology of the disease is still obscure, but is probably a toxemia or a specific microorganism which invades the blood and is essentially identical with septic hemorrhage seen in the newborn (*q. v.*).

Purpura is most frequently observed in children (male and female) over five years of age, and more rarely in younger ones. It occurs either as a primary affection, or in connection with acute infectious diseases, such as scarlatina, measles, typhoid, influenza, etc., and shows a predilection for poorly nourished, anemic and rachitic children living in dark, damp dwellings, with bad hygienic surroundings.

Consonant with the degree of severity of the affection, it is customary to distinguish the following forms of purpura:

1. Purpura Simplex.—The hemorrhage is confined to the skin only, and appears as pinhead- to lentil-sized spots at first upon the lower extremities, but later also on the other portions of the body. Aside from occasional prodromata consisting of gastroenteric disturbance of brief duration, it is free from constitutional manifestations. The majority of these cases pursue a favorable course. The petechiæ either subside entirely within from one week to one month, or return at short or long intervals, in which latter event transition into a severe type of the disease is not uncommon.

2. Purpura s. Peliosis Rheumatica.—(See p. 422.)

3. Purpura Hemorrhagica (Morbus Maculosus Werlhofii).—This form of purpura is manifested by hemorrhages in the skin as well as in the mucous membranes. Its onset is either sudden or preceded by slight prodromata or purpura simplex. The skin petechiæ may vary in size from a lentil to the palm of a hand, and do not disappear on pressure. They usually spread rapidly over the entire body. The hemorrhages into the mucous membranes are rarely very profuse. As a rule, there are only ecchymoses upon the mucous membranes of the nose, gums, and pharynx, but in severe cases the hemorrhagic tendency may extend to almost every structure and organ of the body, so that the patient bleeds from the nose, mouth, ears, retina and choroid, throat, lungs, stomach, bowels, kidneys, genitalia, etc., and sometimes even into the brain and cord. Under these conditions there are well-marked constitutional symptoms (prostration, headache and articular pain, cerebral symptoms as a result of the anemia or meningeal hemorrhage, colic and tenesmus, etc), but in mild cases the patient may appear perfectly well. The course of the disease, therefore, varies with the seat and amount of the bleeding. An attack of purpura hemorrhagica of medium severity usually lasts from ten to fourteen days. After about a week the cutaneous ecchymoses begin to change from the original red to bluish, yellow, greenish and brown, and disappear entirely within another week. The hemorrhages from the mucous membranes and viscera also gradually cease, the general condition of the patient improves, and recovery ensues, apparently

without any serious consequences. On the other hand, in a great many cases, the course of the first attack may be protracted for weeks and months by frequent recurrence of the bleeding, and lead to profound anemia and death, or establish a tendency to relapses, which may manifest themselves on slight provocation.

The blood changes vary with the degree of the hemorrhage. We usually find the usual manifestations of profound anemia.

4. Purpura Fulminans (Henoch).—This type of purpura is essentially identical with the former variety, except that its course is extremely rapid and violent, with severe constitutional symptoms, such as chills, vomiting, intense abdominal pain and intestinal hemorrhage, hyperpyrexia, cerebral symptoms, and collapse. It is invariably fatal, death taking place with symptoms of cardiac paralysis, within from one to four days. Postmortem findings resemble those of severe anemia.

Purpura may occasionally be complicated by gangrene of the skin, subcutaneous tissue or mucous membranes, rendering the prognosis very much worse.

In the early stage of the disease hemorrhea acquista may be mistaken for hemorrhea congenita, infantile scurvy, and exanthemata (scarlatina, morbilli diphtheria, variola, typhoid, etc.) with hemorrhagic symptoms.

Differential Diagnosis

Hemorrhea congenita presents a history of an hereditary tendency, most frequently follows some local injury, and if it occurs spontaneously, it very rarely involves several portions of the body simultaneously.

Infantile scurvy is an affection principally of early infancy and associated with malnutrition. The hemorrhage is also deepseated (subperiosteal).

Exanthemata have pathognomonic symptoms of their own (high fever) which are wanting in purpura. The concurrence of the former with the latter, however, should not be lost sight of.

Septic purpura can readily be recognized by the other septic symptoms.

Treatment.—The treatment of purpura is very unsatisfactory. Mild cases usually recover spontaneously, and grave ones may go from bad to worse even under the best mode of treatment. In bad cases transfusion is indicated. Absolute rest in bed, nutritious diet, plenty of fresh air, iron and arsenic, and the administration of fresh fruit juice will enhance the arrest of milder forms of the disease.

Local hemorrhage should be treated in accordance with the rules laid down for the management of bleeding from other causes (com-

pression, ice bags, styptics, etc). After cessation of the bleeding tonics are useful. Stimulants, in collapse.

Morbus Addisonii

(BRONZED SKIN)

The pathogenesis of this affection is as yet awaiting correct interpretation. While in the majority of cases postmortem examination reveals disease of the suprarenals (caseation or calcification), cases of Addison's disease are also on record which failed to show distinct pathologic change in these glands. The disease usually attacks children over ten years of age, and, exceptionally, younger ones. It is manifested by progressive emaciation, dyspepsia, uncontrollable diarrhea, anemia, and bronze-like discoloration of the skin and mucous membranes. The discoloration begins at the breast nipples, axillary regions, hands and face, and gradually affects the entire body (except the conjunctivæ and nails). The patients succumb within a few months or years to exhaustion and paralysis of the heart.

Hematinics, roborants, and the thymus, suprarenal, parathyroid and pituitary extracts, are deserving of trial.

Diseases of the Spleen

Spleen affections are manifested principally by enlargement of the organ, demonstrable by palpation and percussion.

Movable Spleen

(WANDERING SPLEEN, LIEN MOBILIS)

This condition is important chiefly from a diagnostic point of view, as it is apt to be mistaken for splenic enlargement. It differs from the latter by the absence of constitutional symptoms and by the softer consistence of the spleen. It is usually associated with general atony of the entire musculature, especially of the abdominal wall, and in older children not rarely with enteroptosis, floating liver and kidneys. Subjective symptoms may be absent. Older children may complain of a feeling of weight or pain in the left side, colic, and nausea.

Mild cases frequently obtain permanent relief from the use of an abdominal binder and general tonic treatment (massage, cod liver oil, arsenic). In very pronounced cases surgical interference is indicated.

Acute Splenitis

(Splenic Congestion)

An acute splenic enlargement may be caused by malaria, typhoid, recurrent fever and miliary tuberculosis, more rarely by influenza, rötheln, scarlet fever, tuberculous meningitis, mumps, erysipelas and angina. Very rapid and intense enlargement of the spleen may occasionally be followed by rupture of the spleen, hemorrhage in the abdominal cavity and death.

In the majority of instances the splenitis subsides spontaneously with the underlying cause. If the disease is due to direct infection by pyogenic microorganisms, trauma (with open wound) or metastasis, it may end in suppuration (splenic abscess). Occasionally the inflammation extends to the surrounding tissues, especially to the capsule of the organ, *perisplenitis,* and gives rise to inflammatory adhesions to neighboring structures (diaphragm, colon, or fundus ventriculi).

Chronic Inflammation of the Spleen

(Chronic Hypertrophy, Splenomegaly)

Occasionally chronic enlargement of the spleen is the result of acute splenitis. Most frequently, however, it develops insidiously in connection with chronic malaria, syphilis, tuberculosis, rachitis, leukemia, pseudoleukemia and amyloid degeneration.

The symptoms vary with the original cause and the degree of pressure exerted by the spleen upon the neighboring structures. No attempt will therefore be made to go into a detailed description of the symptomatology. Mention may here be made of the fact that in the so-called "idiopathic" splenomegaly the patient may appear entirely free from constitutional manifestation.

Treatment.—This is symptomatic. If the spleen alone is involved and gives rise to grave pressure symptoms, splenectomy may have to be resorted to.

Banti's Disease

This disease is infrequently observed in children. In some cases a history of syphilis is obtainable. It is characterized by splenomegaly, anemia, ascites, cirrhosis of the liver, and hemorrhages. Postmortem examination usually discloses a fibrosis of the reticulum of the spleen, liver and the portal vein. The bone marrow and lymph nodes are normal. Early splenectomy is said to cure the affection. The diagnosis can be made only by exclusion of similar spleen and liver diseases.

Primary Family Splenomegaly (Gaucher)

This peculiar, apparently congenital, enlargement of the spleen is occasionally (only 4 cases came under my personal observation) encountered in two or more members of the same family. Although accurately described by P. C. E. Gaucher in 1882 (De l' epithélioma primitif, etc., etc.) and carefully studied since then in the living and

Fig. 157.   Fig. 158.

Figs. 157 and 158.—Primary family splenohepatomegaly, Gaucher type, in brother and sister.

postmortem, its etiology is still shrouded in mystery. It is generally overlooked in early infancy, or the splenic and hepatic enlargement is attributed to rachitis, splenic anemia or syphilis. As the child grows older, it is found that notwithstanding good hygienic care and treatment, the affected organs assume greater dimensions, often occupying the entire abdominal cavity. (Fig. 157.) In addition to this

symptom the patient usually suffers from anemia, and its accompanying manifestations; occasional hemorrhage from the nose and mouth; pigmentation of the skin, and enlargement of the lymph nodes. The disease usually proceeds a chronic course and is sometimes marked by remissions or even spontaneous arrest of further enlargement. In the majority of instances, however, death supervenes within from two to ten years as a result of passive congestion of the different adjacent organs which are displaced and pressed upon by the ever-growing spleen and liver.

A correct diagnosis can most frequently be made by excluding syphilis (Wassermann reaction positive), tuberculosis (tuberculin test positive), splenic anemia (definite blood changes, liver usually free), and Banti's disease (spleen but moderately enlarged, usually ascites, not congenital, nor a family affection).

Early splenectomy is the only procedure that offers any prospect of recovery. Cases in which the splenic enlargement progresses very slowly are best let alone.

Postmortem examination usually discloses an endothelial hyperplasia in the spleen, liver, lymph nodes and bone marrow. To give a definite idea of the enormity of the splenic enlargement in this affection I may add that in a case of a thirteen-year-old girl reported by Bovaird (Am. Jour. Med. Sc., 1900), the spleen weighed 12½ pounds.

Adenitis and Lymphadenitis

Acute and chronic involvement of the lymph glands are of quite common occurrence in children. We may classify them in accord with their etiology as follows:*

Inflammations

Acute lymphadenitis due to local infections
- (a) Trauma and skin diseases
- (b) Nasopharyngeal disease
- (c) Dental caries and stomatitis
- (d) Acute infectious diseases, more particularly glandular fever, German measles

Chronic lymphadenitis
- (a) Hyperplasia following acute lymphadenitis
- (b) Syphilis
- (c) Tuberculosis (scrofulosis)
- (d) Leukemia and pseudoleukemia

Tumors
- Lymphosarcoma
- Malignant lymphoma
- Chloroma
- Secondary malignant tumors { Carcinoma / Sarcoma

*Diagram modified after A. Caillé.

The glands most frequently affected are the cervical, submaxillary, submental, axillary, peribronchial, mesenteric and inguinal. Ordinarily with removal of the cause, the glandular enlargement gradually disappears; in a number of cases, however, they remain permanently indurated.

1. Diseases of the ear (auditory meatus), eruptions above the face, and occasionally during parotiditis.

2. Mastoiditis and infections, and eruptions affecting the scalp.

3. Infections of the chin, tongue and lower lip.

4. Infections of the mouth and teeth, stomatitis, rubeola and rubella.

5. Infections of the tonsils, in the mild attacks of scarlet fever and at first in variola. In severe scarlet fever 5, 6, 7 and 8 may be much affected.
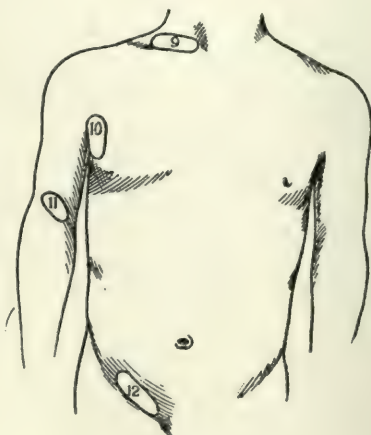
Fig. 159.   Fig. 160.

Figs. 159 and 160.—Distribution of the principal lymphatic glands of the neck and trunk.

6. Pharyngeal infections and inflammation, therefore in retropharyngeal lymphadenitis. Also in severe scarlet fever and rubella.

7. Infections of the scalp and scarlet fever.

8. During the course of diphtheria, 4, 5, 6, 7 and 8 may become prominently enlarged, so that the whole neck appears badly swollen and tender.

9. Infections of the neck and occasionally during the course of diphtheria.

10. Infections affecting the arm, the axilla and the upper portions of the chest anteriorly and posteriorly.

11. Infections of the hand, and especially of the three inner fingers; quite frequently this is enlarged during the course of a syphilitic eruption.

12. Infections affecting the lower limbs, particularly the thigh and sometimes during the course of syphilis. In rare instances these glands are affected in rubella.

Treatment.—Attention to primary cause; ice bag to the swelling or an ointment of plumbum iodide or ichthyol. If the swelling persists,

hot compresses to hasten suppuration, incision and drainage. Tuber-
culous glands are nowadays let alone until they show signs of break-
ing down. They are then incised and drained. In some of these cases
tuberculin treatment (*q. v.*) seems to do good. Glands that give rise
to persistent constitutional symptoms should be removed. Syrup of
iodide of iron and cod liver oil often act very beneficially.

DISEASES OF THE THYROID GLAND

The normal thyroid gland is somewhat larger in children, especially
girls, than in adults. It consists of three lobes, one middle small
lobe (inconstant) and two larger lateral lobes. The latter are con-
nected by an isthmus. The lateral lobes are situated on each side of
the trachea along the second and third tracheal rings; the middle lobe
lies in front of the thyroid cartilage and ascends upward in the direc-
tion of the middle of the hyoid bone. As the gland is thin and often
lies deeply imbedded in the neck, it is very rarely possible by palpa-
tion to determine the size of a normal thyroid.

Thyroiditis

(STRUMITIS)

Primary inflammation of the thyroid gland is usually of traumatic
origin (direct violence, or injury during delivery). It is of very rare
occurrence. More frequently we meet with secondary thyroiditis, as
a rule, in connection with acute exanthematous diseases and occasion-
ally with parotitis, malaria, and articular rheumatism.

The symptomatology consists of swelling of the gland, pain on
pressure as well as on moving the neck, and in some cases redness,
fluctuation and suppuration, and more or less marked pressure symp-
toms.

The inflammation usually disappears under local application of
cold. Should an abscess form, it demands immediate evacuation of
the pus and drainage.

Severe protracted thyroiditis not rarely leads to atrophy of the
gland.

Goiter

(STRUMA)

As in adults, the thyroid gland of children is subject to hyperplasia
and cystic degeneration. In countries where goiter is endemic it is
not rarely observed in very young infants, and is probably of ante-

natal origin. On the other hand, sporadic goiter, as a rule, develops at the period of puberty, particularly in girls.

Small goiters may remain free from any manifestations, except the local swelling in the anterior portion of the neck, while goiters large enough to exert pressure upon the adjacent structures may prove a menace to life by compression of the trachea, and the large blood vessels and nerves which abound in the neck. The pressure symptoms ordinarily consist of headache, dizziness, aphonia, dyspnea and paroxysmal cough. This grave symptom complex, however, is of unusual occurrence.

On the whole, the prognosis is favorable. The great majority of cases of goiter yield promptly to internal administration of small

Fig. 161.—Goiter in girl eleven years old.

doses of iodine, with or without thyroid or parathyroid gland substance, and external use of iodine ointment. Large goiters, causing marked pressure symptoms, call for their extirpation.

In countries where goiter is endemic its development to a great extent may be prevented by change of residence, by boiling the drinking water, and by drinking large quantities of distilled water.

In infants goiter may be mistaken for a large hygroma cysticum colli congenitum or other cysts of the neck, and in older children for exophthalmic goiter. Cysts of the neck are characterized by marked fluctuation and rapid development, and usually start from the submaxillary region.

Exophthalmic Goiter (Basedow's or Graves's Disease)

It is characterized, in addition to the goiter, by tachycardia, muscular tremor, exophthalmos, Graefe's symptom, general ill health, vasomotor disturbances (flushes of the skin alternating with pallor), and pigmentation of the skin. Rapid growth in height is not an uncommon manifestation.

Treatment.—Absolute rest to body and mind. Small doses of atropine to subdue excessive activity of the thyroid. Bromide and digitalis for tachycardia and tremor. Attention to tonsils, teeth and other sources of irritation.

Cretinism

(ENDEMIC OR GOITROUS CRETINISM, SPORADIC CRETINISM
AND MYXIDIOCY)

Cretinism is due to partial or total arrest of the secretion of the thyroid gland, in consequence of congenital or acquired (extirpation) absence, atrophy (from strumitis, syphilis, tuberculosis, or neoplasms), or goitrous degeneration of the gland.

Fig. 162.—Hypothyroidism—Myxidiocy, in a girl sixteen years old.

Endemic cretinism occurs in children living in countries where goiter is endemic, or in descendants of people coming from these regions, and is very frequently associated with goiter. On the other hand, sporadic cretinism is observed in children coming from other parts of the world. The term "myxidiocy" is usually reserved for the pronounced forms of cretinism which are associated with marked pseudolipomatosis. (For full description of "Cretinism," see p. 721.)

DISEASES OF THE THYMUS GLAND

The thymus gland consists of two lateral lobes coming in close contact along the middle line. It is situated in the anterior portion
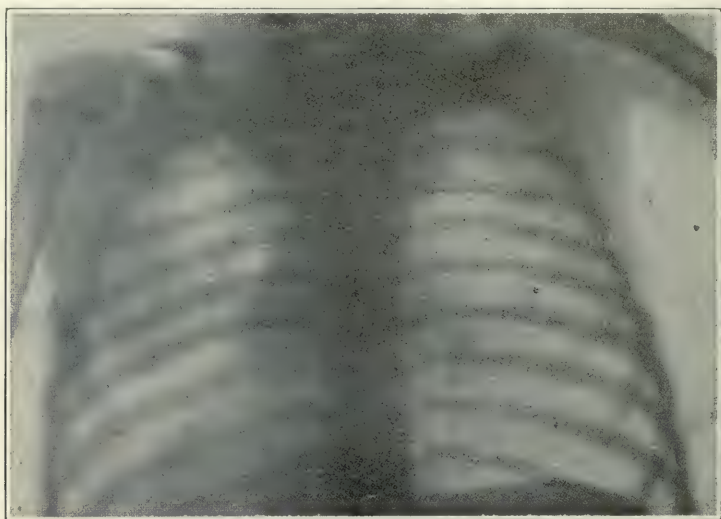
Fig. 163.—Large thymus.

of the neck and superior mediastinum, extending from the lower border of the thyroid gland to the upper border of the fourth rib. The thymus varies greatly in size and weight. It is about $2\frac{1}{2}$ inches in length, $1\frac{1}{2}$ inches in width (at its lower portion), and $\frac{1}{4}$ of an inch in thickness. It attains its greatest development (weighing $\frac{3}{4}$ ounce) between the first and second years, and undergoes rapid degeneration soon after puberty, so that, at the age of twenty, it is a mere vestige of lymphoid tissue and fat. In children under six years of age, light percussion over the superior mediastinum reveals a triangular field of dulness, its base being on a line with the sternoclavicular articulations, and its apex

the second rib. It is well to remember, however, that similar dulness is obtained in enlarged bronchial glands.

We have yet a great deal to learn about the status of the thymus gland in the human economy. Though fully dispensable in adult life, it is essential to the growth and development of the child. The function of the thymus is more or less dependent upon and in part regulated by that of the thyroid, and *vice versa*. Thus, there is less need of thymus when the thyroid is gone, and similarly less thyroid suffices when the thymus is removed. Moreover in cases of hypertrophy of the thymus, where the elaboration of its secretion is excessive, removal of the thyroid is usually followed by sudden death, which shows plainly that in the absence of the regulating power of the thyroid an excess of thymus secretion is destructive to the human economy. Experiments on dogs by H. Klose* have shown that about two weeks after total removal of the thymus gland a "stadium adipositas" of from two to three months' duration develops, followed by loss in weight, general bodily weakness, frailty of the bones, arrest of growth in spite of ravenous appetite, and frequently spontaneous fractures. This cachectic state, or *cachexia thymopriva* is accompanied by *idiotia thymopriva* with a terminal *coma thymicum*. Postmortem examination discloses signs of rachitis, osteomalacia and osteoporosis, the lime salts content being reduced to about half its normal percentage, although the ratio of the lime salts was unaltered. According to the same author the deficiency in undissolved lime salts is dependent upon an increased acid action owing to the failure of the thymus to inhibit the formation of acids in the organism or to neutralize or mask an excess thereof. This "hypothetic" nucleinic acid intoxication, he believes, produces in growing bone defective construction, rachitis with abnormal softness and flexibility, while in fully formed bone increased destruction, osteomalacia, and osteoporosis with abnormal fragility.

With these laboratory observations in view we are enabled to obtain a clearer conception of the manifestations accruing from the effects of thymus disease or congenital abnormality, be it hyperplasia or hypoplasia of the thymus.

Like other glands of the body the thymus gland is subject to acute and chronic inflammation (thymitis) with consecutive hyperplasia, or premature atrophy; tuberculosis; syphilis, and neoplasms.

*Brooks and Langerhans' "Text-Book of Pathology."

Acute Thymitis

Acute thymitis is a rare affection, generally occurring only in young infants. The etiology is obscure: in some cases a history of naso-pharyngeal infection is elicited. It is also claimed that thymitis may develop secondarily to a systemic pyemic process or by extension of inflammation from adjacent structures. The symptomatology is usually indefinite, except where the thymus attains an enormous size and gives rise to pressure symptoms on the larnyx, esophagus, and the large blood vessels in the mediastinum, etc. The case presently to be related may offer a general idea of the puzzling symptom complex. The baby was four weeks old, normal at birth and nursed by the mother. It was circumcised when eight days old, and did well very soon after. The day before coming under my observation it was very restless, and towards evening, while at the breast, suddenly dropped the nipple, gave a sharp loud cry, snapped the jaws tightly together, and seemed to lose consciousness. A neighboring physician who was immediately consulted, did not venture to express a definite opinion, but prescribed calomel and an intestinal irrigation. The next day I found the baby in the following condition. The baby looked well nourished and otherwise normal in appearance. It lay perfectly still with its eyes widely open. There was neither rigidity of the neck, nor any other sign (Brudzinski, Kernig, etc.) of disease of the nervous system. The respirations ranged between 70 and 80 per minute, and were acompanied by an expiratory moan. The heart beat ranged between 120 to 130 per minute. Both the lungs and heart were absolutely free from any abnormal physical signs. The liver and spleen were normal in size. The urine although greatly suppressed in the beginning, showed no abnormal constituents. There was relative constipation, but the bowels moved with enema. The abdomen was soft and free from any tumefaction. The blood was negative except for a high leucocytosis. The temperature was below 100° F., but rose to 102° and 103° F. a few days later. The jaws remained somewhat rigid, and the baby refused to nurse at the breast. He swallowed milk dropped in his mouth, apparently with ease, in the first three or four days of his illness, but with difficulty towards the end. At this time attacks of asphyxia and continued cyanosis also set in. The baby was ill six days and died without a struggle. The two learned pediatrists whom I consulted on the case, like myself, failed to arrive at a correct diagnosis. We all suspected a toxic condition of the blood, but never thought of the thymus gland. Some time later I had the opportunity to discuss the case with Dr. Charles G. Kerley, who

kindly informed me that he had seen a few cases of the kind at the Babies' Hospital and that postmortem examination disclosed acute inflammation of the thymus gland. I firmly believe we were dealing with just such a case.

Acute thymitis sometimes leads to suppuration of the gland.

Chronic Thymitis

Chronic thymus disease is variously attributed to lung and heart disease, pertussis, rubeola, diphtheria, scarlatina, asphyxia neonatorum, tuberculous and syphilitic processes, and malignant growths, but excepting the last three etiologic factors, the manifestations produced by the other causes are undoubtedly only transient in character. The same is true of atrophy of the thymus associated with protracted malnutrition. Indeed, it has been shown that the atrophied gland in emaciation is often regenerated on restoration of the body weight. I am inclined to believe, therefore, that genuine hypo- or hyperplasia of the thymus is due either to a primary congenital anomaly of the thymus, or develops later secondarily to alterations in the thyroid or other lymphatic gland.

Whatever the cause, the clinical syndromes arising from the effects of hypo- or hyperplasia of the thymus are not as difficult of recognition as was formerly believed. As already stated the functions of the thymus and thyroid glands are closely correlated, so that hypoplasia of the thymus by inducing also a reciprocal diminution in the thyroid secretion produces not only an arrest of growth and frailty of the bones and general debility, but also lowered mental capacity as exemplified in infantilism, more especially in the Brissaud and Lorain types.

The clinical signs of hyperplasia differ with the degree of the thymic enlargement and the functional activity of the thymus. Not every case of thymus hypertrophy is necessarily associated with increased function of the gland. The latter may, for example, owe its enlargement to a growth destroying the medullary substance of the gland, and thus be incapacitated rather than hyperactivated. Even where the thymus secretion is excessive, the symptoms engendered vary greatly. By corresponding increased activity of the thyroid the clinical picture may be limited to that observed in hyperthyroidism, i. e., tachycardia, insomnia, change in disposition, abnormal perspiration, and often also a mild degree of exophthalmos and possibly struma.

In another group of cases of hyperplasia of the thymus careful examination of the child fails to reveal any definite manifestations of a pathologic entity, except possibly a few signs of anemia with

adipositas, rachitis, or "scrofulosis," but this notwithstanding, death may occur without any apparent reason as the result of slight causes (*e. g.*, serum injection, narcosis, slight operations, etc.) which produce physical excitement or shock and are usually of no consequence in healthy children. This condition is generally spoken of as *status lymphaticus* or lymphatism. Unfortunately, we have no characteristic symptoms by means of which this condition may be diagnosed during life and nothing characteristic is found at necropsy.

Finally, in another group of cases hyperplasia of the thymus percussion reveals marked dulness over the upper portion of the sternum particularly to the left as low as the second rib and often also to the back between the scapulæ. It is in addition distinguished by the presence of swollen lymph glands in the lateral lower region of the neck, which may sometimes be seen to continue deeply down between the clavicle and side of the sternum. If the hypertrophy is of long standing we readily detect secondary manifestations, such as dilatation of the veins of the neck, dislocation of the heart, accentuation of auscultatory signs of the heart and lungs and arching and distention of the thorax. The thymus gland may occasionally be felt in the middle line above the incisura sterni as an arched elastic swelling, which may ascend upward to the thyroid gland. Furthermore, the thymus enlargement is distinctly discernible by means of the Roentgen rays. The clinical signs differ, of course, with the degree of mechanical encroachment upon the adjacent structures (thyroid, blood vessels and nerves, trachea and bronchi) and secondary involvement of the heart and lungs. As a rule, the symptomatology is essentially that of cardiac asthma, so-called, and is generally spoken of as *asthma thymicum*. The child is suffering from a persistent cough and other signs of bronchitis, marked dyspnea, cyanosis or marked pallor of the face and, off and on, with acute turgescence of the enlarged thymus, it is attacked by paroxysms of asphyxia which not rarely terminate fatally. Pott,* who has frequently observed the course of these fatal attacks of asphyxia, describes them as follows. The children bend their heads suddenly backward (which position, by the way, by producing a marked lordosis of the cervical region of the spine and thus increasing the pressure of the thymus against the trachea greatly aggravates the laryngospasm), and make soundless, gasping inspiratory movements. The eyes are turned, the face is blue or black, the cyanotic tongue is impacted between the jaws, the veins of the neck are distended, the hands are clinched, the forearms are pronated and abducted, the legs are stiff and extended, the large toes are

---

*Graetzer and Sheffield's Practical Pediatrics, p. 296.

abducted and flexed, and the spine is arched strongly backward. The pulse, heart's action, and heart sounds cease immediately with the onset of the paroxysm, and after a few futile respiratory efforts the face turns ashy in color and in a minute or two the child is a corpse. Pott believes to have felt the vocal cords snugly together in the median line. He nevertheless maintains that death is caused by heart failure and not by closure of the glottis, for on two occasions he performed immediate tracheotomy by *one* incision without any relief. This view is not shared by all observers, and Biedert, for example, is of the opinion that closure of the glottis, through suffocation stasis in the heart and thymus might be responsible for the onset of such an attack and its grave result. Of course, not all attacks terminate immediately fatally. Some time ago I had the opportunity, for several weeks, to watch a five-months-old infant afflicted with an apparently congenital hyperplasia of the thymus. The family history was negative as regards syphilis and tuberculosis. The father, a French artist, was in perfect health, the mother was subject to arthritis with slight valvular heart disease. The older child, who is now ten years old, was for four months suffering from spasmodic pyloric stenosis. The infant under consideration weighed about 5 pounds at birth and failed to gain though breast-fed for the first three months of its life. It was noticed immediately after birth that he was very pale and cyanotic, had some difficulty in breathing, coughed a little and had a husky cry. Gradually these symptoms became worse and attacks of bronchitis with marked dyspnea set in, during which the baby would repeatedly be seized by convulsions with loss of consciousness. The family physician diagnosed it as asthma, while a very learned pediatrist decided that they were dealing with spasmus glottidis supervening faulty feeding. The child's condition got gradually worse, notwithstanding the correction of diet and administration of antispasmodics, etc. When I saw it the clinical picture resembled pulmonary edema, but there were no signs of primary heart or kidney disease and the history of the case and the distinct hoarseness certainly pointed to some form of immediate obstruction in the upper respiratory tract—most probably primary, nay congenital, since it was manifested at birth. There was no difficulty to exclude asthma, as this disease is invariably associated with remissions and would at least temporarily yield to antiasthmatic remedies which in this case were given a fair trial. Neither did the diagnosis of spasmus glottidis appeal to me. Here the infant is free from dyspnea, etc., between each paroxysm, and certainly does not get worse on a suitable diet. Going over the case more carefully I

detected undue dulness over the upper portion of the sternum, marked dilatation of the veins in the neck, fulness of the neck over the interclavicular notch—all signs and symptoms which were quite suggestive of thymus hypertrophy. I therefore suggested a Roentgen-ray examination, which promptly confirmed my diagnosis. The infant was in too delicate a condition to undergo thymectomy, and his parents—rightly—hesitated to consent to this procedure. The infant was soon relieved of its agony during a paroxysm of asphyxia.

Treatment.—Acute thymitis, if detected early may occasionally be arrested in its progress by an ice bag, and in strong infants a few

Fig. 164.—Precocious child eight years old; began to menstruate when about five years old (hyperpituitaria?).

leeches over the manubrium sterni, and internal administration of calomel. In chronic thymus hypertrophy, if severe in character, partial or even complete removal of the thymus is absolutely indispensable. Before resorting to an operation, however, we must ascertain that we are not dealing with a syphilitic condition, which may be remedied by antisyphilitic medication. Sudden attacks of asphyxia calls for prompt tracheotomy or intubation and antispas-

modies (bromides, codeine). In hypoplasia of the thymus, some bene-
fit may possibly be derived from internal administration of thymus
gland, from 10 to 30 grains daily.

Disease of the Pituitary Gland, or Hypophysis Cerebri

(HYPERPITUITARIA; HYPOPITUITARIA; DYSTROPHIA ADIPOSOGENITALIS,
FRÖHLICH)

The pituitary body consists of an anterior, middle and posterior
portion, and is situated at the base of the brain, resting upon and
slightly surrounded by the sella turcica. Disturbances in the function
of the anterior portion of the hypophysis give rise to gigantism in
children and acromegaly in adults, while disturbances in the function
of the posterior (or both anterior and posterior) portion of the hypo-
physis leads to dystrophia adiposogenitalis, a clinical syndrome first
described by A. Fröhlich in 1911. This clinical entity is manifested by
retarded growth and development, excessive adipositas, sexual in-
fantilism, atrophy of the sexual glands, polyuria, subnormal tempera-
ture, sluggish metabolism, and high tolerance for carbohydrates (ex-
cessive amounts of sugar fail to produce glycosuria).

In treating pituitary disease it is essential first of all to determine
by exact Roentgen-ray examinations, whether or not a hypophyseal
tumor is being dealt with. If this be so, operative interference is
indispensable, and is nowadays executed with a fair amount of suc-
cess. In the absence of a tumor, pituitary feeding should be resorted
to, particularly in cases of hypo- and apituitarism.

DISEASES OF THE KIDNEYS, BLADDER, ETC.

Nephritis Acuta

Acute nephritis is most frequently met with in association with acute infectious and contagious diseases, especially scarlatina, diphtheria, tonsillitis, influenza, and pneumonia. Less frequently it occurs as a result of exposure to wet and cold; of structural alterations of the skin, *e. g.*, extensive burns; of ingestion of certain irritants to the kidneys, *e. g.*, cantharides, potassium chlorate, aspidium, etc., ether or chloroform inhalation, and, finally, not rarely it is observed in infants suffering from gastroenteric affections. The aforementioned causes usually operate upon both kidneys, so that both kidneys are equally affected. The lesion may, however, remain limited to one kidney where the disease is caused by direct, unilateral trauma (direct violence, calculus, etc.). The seat of the kidney lesions varies somewhat with the cause. For example, the glomeruli (*glomerular nephritis*) are most severely involved in scarlatina, while in diphtheria we most commonly find degenerative changes in the renal tubules (*degenerative or parenchymatous nephritis*). But no particular form of acute nephritis is peculiar to a given cause. In severe cases the kidneys are greatly increased in volume and weight. The surface is smooth and the capsule readily removable. The renal cortex is either uniformly reddened or pale and mottled with red. The tubuli uriniferi are partly or completely obstructed by large granular epithelial cells, blood corpuscles and fibrin. In the early stage of the disease the interstitial tissue shows no alteration; in protracted cases, however, this tissue may suffer very severely. In this event the process is often spoken of as productive or *interstitial nephritis*.

Consonant with the etiologic factors we distinguish a primary and secondary form of acute nephritis, but, except for some slight difference in the onset (it being more sudden in primary nephritis), the symptomatology is practically the same in both varieties. The child complains of headache, backache, dizziness, nausea and chilliness, occasionally vomits, and in severe forms shows other symptoms of grave constitutional disturbance. Not infrequently attention to the illness is not attracted until the appearance of pronounced anemia

and puffiness of the eyelids, or, especially in infants, the occurrence of partial or total suppression of urine with or without uremic symptoms. Examination of the urine discloses more or less marked alteration in its constituents. Chemically, the urine almost invariably reveals the presence of a variable amount of albumin, and, microscopically, casts of all sorts, especially hyaline, red and white blood corpuscles, epithelium, detritus, etc. The urine is usually acid, and its specific gravity high, the latter being, of course, most marked when the quantity is very small. The excretion of urea is diminished. In

Fig. 165.—Acute nephritis with general anasarca in a four-month-old infant. Note "pitting" on pressure with finger.

Fig. 166.—Same case as Fig. 165 three weeks later.

severe inflammation of the kidneys the urine contains a large quantity of blood (*hemorrhagic nephritis*), and is dark-red or smoky in color.

As already alluded to, the onset of nephritis often escapes detection. This is especially true in the secondary form. Hence the importance of systematic examination of the urine during the course of acute communicable diseases. It is well to remember, however, that not every albuminuria is of nephritic origin. A small quantity of albumin and a few casts are not rarely found in acute febrile diseases (*e. g.*,

PATHOLOGIC URINARY FINDINGS†

Disease	Quantity	Reaction	Specific Gravity	Color	Total Solids	Urinary Sediment	Blood	Albumin	Chlorids	Urea	Dropsy	Microscopic Findings
Parenchymatous Nephritis (Acute)	5 to 500 c.c.	Acid	1020-1030	Smoky	Diminished	Abundant	Present	1 to 2%	Absent or rarely present	Diminished	Present (Onset sudden)	Pus cells, blood cells, epithelial casts, hyaline casts, blood casts
Parenchymatous Nephritis (Chronic)	10 to 1000 c.c.	Acid	1020-1025	Dark	Diminished	Considerable	Absent	1 to 5%	Normal	May be absent or diminished	Marked	Granular casts and cells. Fatty casts, fat cells, free fat
Interstitial Nephritis (Chronic)	25 to 2000 c.c.	Acid	1008-1010	Pale	Diminished	Slight	Absent	Slight or trace	Diminished	Diminished	Rarely marked	Pale granular casts, hyaline casts
Diabetes Mellitus	500 to 10000 c.c.	Acid	1030-1040	Pale or greenish	Increased	None	Absent	absent* or trace	Increased	Increased	Absent	Renal epithelial cells, oxalate of calcium
Diabetes Insipidus	6000 to 10000 c.c.	Acid or neutral	1002-1006	Pale	Increased	None	Absent	Trace or absent	Increased	Increased	Absent. edema late	Earthly phosphates
Acute Pyelitis	Diminished	Acid	High	Dark	About Normal	Abundant	Present	Trace	Diminished	Normal or diminished	Absent	Uric acid, oxalate of calcium, blood cells, pus cells
Chronic Pyelitis	Diminished	Acid or alkaline	1010-1015	Pale	Diminished	Abundant	Rarely present	Present	Diminished	Diminished	Absent	Pus cells. Sometimes blood cells
Acute Cystitis	Scanty	Acid becoming alkaline	High	Cloudy or bloody	Normal	Considerable	Present	Trace	Diminished	Normal	Absent	Round epithelial cells, squamous epithelial cells, pus cells, blood cells
Chronic Cystitis	Normal	Alkaline	Lowered	Turbid	Diminished	Considerable	May be present	Trace	Normal	Diminished	Absent	Epithelial cells, pus cells, triple phosphates, crystals

*Glycosuria.

†Modified after Heitzman.

in the beginning of scarlatina) without kidney lesions and are only transitory in nature.

Cases running a favorable course begin markedly to improve after about two weeks. The albumin diminishes, the urine increases in quantity, becomes light and clear, and the microscopic abnormal constituents subside. Edema, if present, is slight, and usually limited to the eyelids and rapidly disappears with the improvement of the other symptoms.

Less favorable cases are of longer duration. From day to day the edema assumes wider dimensions, involving the dorsi of the feet, the legs, the genitalia, and, if not checked, the serous effusion may rapidly fill the abdominal and thoracic cavities. In the majority of instances, however, gradual recovery from the immediate attack occurs, although in these cases a relapse must always be apprehended.

Another group of cases is characterized by great diminution of urine (oliguria) or total suppression and consecutive uremia. The latter is manifested by intense headache, dizziness, vomiting, dimness of vision up to total blindness, disturbance of hearing, slight twitching up to repeated attacks of severe convulsions, slow, irregular pulse, dyspnea, somnolence, sopor and possibly coma and death.

The incipient symptoms of nephritis offer no reliable indications as to the further course of the disease. Scarlatinal nephritis, for example, ushers in with vomiting, intense headache, convulsions, local or general dropsy, and yet often clears up completely within two or three weeks; and, conversely, nephritis may set in insidiously, apparently free from any alarming symptoms, and, nevertheless, proceed a very protracted course and possibly lead to permanent degeneration of the kidney structures. Furthermore, relapses may complicate matters, often when recovery is imminent.

The prognosis, therefore, should always be guarded, even though the general condition of the patient is good. Even in mild cases untoward complications are apt to supervene. Serous effusions in internal cavities are not rare. This is true especially of ascites, less frequently of pleural or pericardial effusions. The heart rarely escapes involvement. Hypertrophy of the heart is quite common, and, if the nephritis runs a protracted course, dilatation of the heart may prove a very dangerous complication, particularly in view of the secondary pulmonary edema, which is very prone to occur in such cases, and often proves fatal. Extensive anasarca with scanty urine, especially if ascites is associated with hydrothorax, greatly mars the prognosis. As further complications we may mention uremia, pneumonia, edema of the glottis, severe intestinal catarrh, more rarely

peritonitis, pericarditis and endocarditis (more frequent in scarlatinal nephritis). Notwithstanding, however, the great array of complications, immediate death from acute nephritis, especially the primary variety, is not common. The death rate ranges from between 5 and 20 per cent, the variation depending upon the primary cause, mode of treatment and severity of the complications. A great many patients who survive the acute stage remain invalided for life. As we shall see later, gradual transition from acute into chronic nephritis is not of uncommon occurrence. Convalescence is often prolonged for weeks and months, and even without permanent injury to the kidneys albumin may recur in the urine from time to time for a period of a year or two longer and continue to undermine the child's constitution.

Treatment.—Every case of nephritis, be it ever so mild, should be taken seriously, and kept under strict observation not only during the active stage of the affection, but for many months after. During the acute stage perfect rest in bed should be enjoined and the diet limited to bland articles of food free from salt, preferably milk in moderate quantity with strained oatmeal or barley, zwieback with sweet butter, stale bread with a little apple sauce, and occasionally a little chicken soup. In the absence of edema the drinking of water is not limited, but, otherwise, the partaking of water should be restricted to a few tumblerfuls of Vichy or lithia water per day. As the condition improves the dietary may be augmented by the addition of freshly boiled—without salt—vegetables, such as carrots, spinach, cauliflower, fresh green peas, etc., stewed fruit, and freshly boiled whitefish. The bowels should be kept open by an occasional dose of calomel followed by citrate of magnesia and by daily high intestinal irrigation. Where the excretion of urine is greatly reduced and the dropsy marked, energetic measures should be instituted without delay to relieve the kidney. This should be attempted, not, as is frequently advised, by means of active diuretics, which only help to increase the renal congestion, but by stimulating the activity of the skin and bowels and allaying the irritation of the kidney. For this purpose we resort to hot packs (105° F.), hot baths (103° F.), and hot (110° F.) rectal enemas (to be retained as long as possible) or the Murphy drip. These may be repeated every six hours. Perspiration may be stimulated by small quantities of hot water, or hot lemonade. In hemorrhagic nephritis small doses of ergot act beneficially. Camphor will be found valuable to counteract collapse, and should be administered hypodermically in the form of sterilized camphor-

ated oil. Excessive irritability of the nervous system should be combated by means of the bromides and chloral.

By carefully following the aforementioned directions, uremia is of rare occurrence. Uremic convulsions should be controlled by chloroform inhalation, hypodermic injection of morphine and atropine (for a child two years old $\frac{1}{32}$ grain of morphine and $\frac{1}{500}$ grain of atropine, if necessary to be repeated once after two hours), and where these therapeutic measures fail, by lumbar puncture.

Children recovering from nephritis should not be exposed to the ill effects of overfeeding, overexertion, and exposure to marked atmospheric changes. They should wear light woolen underwear, and, financial means permitting, should spend the winter following an acute attack of nephritis in a warm climate.

To overcome the remaining anemia, iron and cod liver oil will be found of service.

Nephritis Chronica

In the majority of instances chronic nephritis develops as a sequel of the acute affection of the kidneys. The parenchyma or interstitial tissue, or both, remain permanently impaired. On the one hand, we may find the kidneys greatly enlarged, the cortical portion increased in volume, its surface white or pale-yellow—the large white kidney, or parenchymatous nephritis; on the other, the whole organ is reduced in size, the capsule firmly adherent, and the surface irregular and nodular—the granular or cirrhotic kidney, or interstitial nephritis. Amyloid degeneration is another form of chronic nephritis in childhood. It is usually associated with amyloid degeneration of the liver and spleen, and ordinarily occurs secondarily to suppurative processes of the bones or joints. Occasionally chronic nephritis is encountered in connection with congenital malformations of the kidneys, or as a result of hereditary syphilis, tuberculosis, and heart disease.

In the early stages of chronic nephritis the diagnosis rests principally upon the chemic and microscopic findings in the urine. (See p. 574.) In parenchymatous nephritis the quantity of urine is normal or diminished, the specific gravity normal or increased, the albumin content high, and the color cloudy, brownish yellow or bloody. In interstitial nephritis the quantity of urine is increased, the specific gravity low, the albumin content low (occasionally no albumin), and the color clear, and pale. In amyloid degeneration the urine is rich in serum albumin and globulin. Its quantity is often increased. Casts in the urine are present in all varieties.

Where laboratory facilities are at our command, it is of great diag-

nostic and prognostic advantage to determine the functional capacity of the kidneys by means of the phenolsulphonephthalein test and the "two-hour" renal test.

With further advance of the disease, the gradually appearing profound anemia, digestive and respiratory disturbances, local and general dropsy, and cardiac debility readily disclose the underlying condition. Toward the end of life the symptoms resemble greatly those of non-compensating heart disease.

Parenchymatous nephritis offers the worst prognosis, death usually setting in within a year from the appearance of the secondary symptoms. The course of interstitial and amyloid nephritis is much more protracted, and cases of amyloid kidney are on record that markedly improved on removal of the suppurative bone affection; however, complete recovery is practically out of the question.

Treatment.—Under suitable treatment (except in parenchymatous variety) life may be prolonged for many years. As in acute nephritis, the diet should be free from salt, but otherwise should be more liberal. Older children may live on a mixed diet; the following foodstuffs, however, are to be exempt from the list: liver, ham, brains, kidneys, beef-juice, and beef extract, soups, coffee, liquors and spices. All meats, eggs and fish should be taken sparingly. Whenever possible, the child should live in a warm climate. Outdoor life and very *light* exercise are desirable. Daily warm baths with gentle massage act beneficially. With the appearance of dropsy, dyspnea, or other grave symptoms, the patient should be put to bed and treated in the manner outlined under "Acute Nephritis" and "Chronic Heart Disease" (*q. v.*).

Hematinics, in small doses, and other tonics in the form of cod liver oil, nux vomica, and digestants are in order as necessity arises. Excessive dropsical effusions should be relieved by active catharsis, alkaline diuretics, and heart stimulants (digitalis, diuretin), in addition to the therapeutic measures recommended in dropsy accompanying acute nephritis.

In protracted cases, considerable benefit may be derived from Karell's diet, which consists of 60 to 200 c.c. of skimmed milk every four hours during the day, with exclusion of every other food and drink. In some cases protein milk may be tried. Splitting or extirpation of the kidney capsule (Edebohls' operation) will often prolong life.

Nephrolithiasis

(Stones in the Kidney, Renal Calculi)

Renal calculi in children give rise to symptoms identical with those observed in adults. Thus, sudden attacks of pain in the lumbar region, radiating downward along the course of the ureters, groins,

and, in the male, to the testicles. The attacks are usually associated with nausea, vomiting, and sometimes convulsions and collapse. The urine is passed frequently, in small quantities, and contains blood and pus cells. The urine, however, may appear normal if it is excreted from the healthy kidney only; or there may be complete anuria if both ureters are simultaneously obstructed.

Treatment.—Where the stones remain impacted in the ureter, the

Fig. 167.—Oval calculus in left ureter and one just emerging from lower pole of left kidney in a child nine years old.

condition is apt to become very grave in consequence of supervening hydronephrosis, pyonephrosis, or pyelonephritis. In this event we are often obliged to resort to surgical interference. Otherwise symptomatic treatment usually suffices to effect marked improvement or even a cure. Alkalies (piperazine!) should be administered in *uric acid* concrements; sodium phosphate in *oxalic acid,* and citric acid and acetic acid in *phosphatic* concrements. The diet should be bland (avoidance of meat), and metabolism enhanced by digestives, mild laxatives (½ ounce of Margarita water in hot water every morning), moderate exercise, hydrotherapy and massage. To relieve an attack we resort to anodynes (morphine and atrophine hypodermically or a codeine suppository), hot baths and hot poultices.

Right-sided nephrolithiasis may be mistaken for acute appendicitis. But in addition to the pathognomonic signs of appendicitis it will generally be found that the patient suffering from appendicitis keeps perfectly still in bed during the acute stage of the attack, whereas, the nephrolithiasis patient is quite active, moving from place to place while the pain is most severe.

An x-ray examination is often decisive in the diagnosis between nephrolithiasis and appendicitis. In older children cystoscopy will greatly aid in the diagnosis. (See "Uric Acid Infarcts".)

Pyelitis,* Pyelonephritis, Pyelonephrosis

Inflammation of the pelvis of the kidney and contiguous structures with consecutive suppuration usually occurs, as first demonstrated by Escherich, as a result of infection by the Bacillus coli communis (either secondarily to enteric infection, or by systemic infection through some lesion in the intestinal mucosa); as a sequel of infectious diseases, such as scarlatina, diphtheria, variola, or pyemia, or by extension of a suppurative process from the neighboring tissues or organs, *e. g.,* perinephritic abscess, cystitis, colicystitis, (*q. v*), purulent vulvovaginitis and also as a result of direct injury to the lining mucous membrane, *e. g.,* renal stones. It is also met with in connection with congenital malformations of the kidneys or ureters, renal tuberculosis and tumors. The pyelitis may be unilateral (when due to a local cause) or bilateral.

The symptomatology of pyelitis varies greatly with the cause and the course it pursues. In acute cases there are rigors, high and fluctuating temperatures, frequent and scanty urination, pain in the lumbar region (often elicited also on palpation) and, above all, pyuria. The morphologic constituents of the urine vary with the degree of

---

*See also Pyelocystitis.

involvement of the kidneys, ureters and bladder. In a large number of cases the pyelitis is masked by the primary affection and can only be detected by examination of the urine—which should invariably be done where irregular, high fever, without apparent cause, prevails. Cases pursuing a chronic course are ordinarily free from febrile excursions, but the children are pale or waxy in color, complain of headache, lassitude or cardiac palpitation and other symptoms of wasting diseases. Pyonephritis with pus retention (pyonephrosis) often gives rise to a palpable tumor.

Where the cause is removable, and prompt treatment is instituted, the pyelitis may entirely disappear and leave the kidney uninjured. Otherwise the prognosis, as to complete recovery, is bad. The prognosis as to life depends entirely upon the exciting cause and complications, nephritis and exhaustion forming the principal sources of danger.

Treatment.—The aim of the treatment, therefore, should be to avoid nephritis by early elimination of the fundamental disease, and prevention of recurrences of the attacks. The details of such treatment are fully outlined when speaking of the disease in question. Otherwise the treatment is symptomatic. Rest in bed and liquid diet during the acute course of the disease. Urotropin, from 3 to 5 grains every four hours, is indicated in all cases. The urine should be rendered alkaline and as aseptic as possible. This is best accomplished by a liberal supply of water, alkaline diuretics such as potassium citrate (*gr. x t. i. d.*) in addition to the hexamethylenamine. Of late attempts have been made to cure chronic pyelitis by irrigating and draining the renal pelvis. Kretschmer* and Helmholz claim to have cured a number of cases of pyelitis by injecting into the renal pelvis 1 c.c. to 5 c.c. of a 0.5 per cent of silver nitrate solution. The injections may be repeated once or twice, until the urine cultures become sterile. The results as a whole are not very encouraging. The same is true of the administration of vaccines. Pyonephrosis calls for surgical interference. (See also "Pyelocystitis".)

Hemoglobinuria

Hemoglobin or methemoglobin in the urine is occasionally observed in infants and older children, either as a result of poisoning by phosphorus, potassium chlorate, carbolic acid, etc., or in connection with severe burns, acute and chronic infectious diseases, such as exanthemata, malaria, and hereditary syphilis. The urine is mahogany-brown or black in color, greatly resembling bloody urine. Microscopically, however, it shows the presence of blood coloring substance

only, but no blood corpuscles. The spectroscope discloses bands of hemoglobin. The attacks of the hemoglobinuria are of brief duration (sometimes last but a few hours), and are manifested by debility, chilliness, cyanosis, and sometimes high fever. These symptoms disappear as the urine clears up, which ordinarily occurs within a few hours or days. Occasionally the hemoglobinuria appears in paroxysms (*paroxysmal hemoglobinuria*) without any discernible cause or after exposure to cold or undue fatigue.

By rest in bed, liberal supply of liquids, and attention to the exciting cause, the hemoglobinuria subsides without any serious consequences. (See "Epidemic Hemoglobinuria".)

Orthotic, Lordotic, Cyclic or Functional Albuminuria

As the term (orthotic: standing up) indicates, the disease is characterized by the presence of albuminuria after the patient has been up and around (usually several minutes after the erect posture has been assumed) and by its absence while he is perfectly at rest. It is observed especially in delicate children of from five to fifteen years of age, and seems to have nothing in common with organic kidney disease. It has been observed that children suffering from lordosis in the upper lumbar spine are especially prone to be affected by the disease—the spinal deformity by pressure upon the kidneys apparently interfering with the renal circulation. A family predisposition has been traced in some cases, and a history of scarlatina and diphtheria in others. The urine is ordinarily free from abnormal morphologic constituents, the opposite, of course, being the case in true renal disease.

Treatment.—Under suitable treatment, which is essentially the same as in the early stage of chronic nephritis plus correction of lordosis, if there be any, the albuminuria often disappears for a time, but may return after a shorter or longer interval (intermittent form). Notwithstanding the continuance of the albuminuria for many years, the system is very little affected by it, and the prognosis as to life is good. Transition of cyclic albuminuria into nephritis, however, is on record.

Tumors of the Kidney

Aside from tuberculosis and syphilis, which have been discussed elsewhere, the kidneys are occasionally the seat also of benign and malignant neoplasms. The benign tumors (adenoma, fibroma lipoma, cysts, etc.) owing to their very slow growth, generally escape observation, and are often found postmortem in children who during life never

manifested signs of kidney growths. To a great extent this is true also of malignant tumors (sarcoma, carcinoma, myosarcoma, and adenosarcoma) in their early stages of development, since at this period the tumor is barely palpable, and the two additional characteristic signs of malignant kidney growths (*i. e.* hematuria and cachexia) are present only in a small percentage of such cases (usually carcinoma) and are encountered also in a number of other wasting and hemorrhagic diseases. Moreover, hematuria is often absent during the late stage, when the tumor encroaches upon the ureter and obstructs the flow of urine from the affected kidney. Ascites is a frequent symptom, and the colon is usually pushed in front of the tumor. As the growth ad-

Fig. 168.—Adenosarcoma of right kidney in a boy twenty-seven months old, occupying almost the entire abdomen.

vances it spreads in all directions displacing the liver, spleen, heart and lungs, and occupies the entire abdominal cavity. Not rarely, secondary metastases are formed in the other kidney, in the liver, spleen, intestines and retroperitoneal glands, and by pressure upon the ureter, give rise to hydronephrosis. Roentgen-ray examination is helpful in the diagnosis.

Treatment.—Unless operated upon early—which treatment should invariably be recommended—the children usually succumb to progressive emaciation and exhaustion within about a year from the time the tumor makes itself felt. As the majority of the growths are of antenatal origin, nothing can be done in the way of prophylaxis.

Cystitis, Colicystitis

(PYELOCYSTITIS)

Inflammation of the bladder may occur as a primary or secondary disease. Primary cystitis is extremely rare in children, more especially in infants, since the principal cause—direct mechanical injury of the mucous membrane by surgical instruments or other foreign bodies—is but rarely operative in young children. On the other hand, secondary cystitis is of comparatively frequent occurrence, more particularly in girls, and may arise from a great many causes, the most important being infectious diseases (diphtheria, scarlatina, etc.), kidney and bladder disease (calculi, pyelitis, tuberculosis, tumors, etc.), cerebrospinal affections (atony and overdistension of the bladder with consecutive inflammation by decomposed urine), intestinal diseases (invasion of the bladder by the colon bacillus—(*Colicystitis*), and diseases of the vagina and urethra, especially of gonorrheal origin (by extension of the inflammation). Cystitis may follow chemical irritation (from overdoses of cantharides, balsams, liquors, etc.), exposure to cold (sitting on cold stones, etc.), and direct external violence.

The lesions in the bladder may range from simple localized redness to extensive ulceration of the mucous membrane and pseudomembranous deposit. In cases of long standing the inflammation is prone to spread to the ureters and kidneys. In chronic cystitis the mucosa assumes a gray, pigmented color, becomes greatly hypertrophied, and is covered by mucopurulent masses.

In accord with the severity and extent of the lesion cystitis may be manifested by mild or grave symptoms. The latter are most pronounced in primary cases, in those associated with infectious diseases (*e. g.*, diphtheria), and in infection by the colon bacillus. In mild cases the symptomatology consists of painful and frequent micturition, sensitiveness over the region of the bladder, sometimes rectal tenesmus and excoriation of the urethral orifice and of the contiguous structures. The urine is voided in small quantities, sometimes only a few drops at a time, and contains mucous shreds, bladder epithelium, pus corpuscles, blood corpuscles, and numerous bacteria. The urine in simple cystitis is neutral or alkaline, cloudy and dark red, and may contain pieces of membrane, if the cystitis is of diphtheritic origin. On the other hand, in *colicystitis* the urine is acid in reaction, and in addition to the aforementioned constituents we find a large quantity of pus and some albumin, and, not rarely, there are marked constitutional disturbances, such as vomiting, chills, irregular fever, and sometimes convulsions (particularly if anuria exists). The local

symptoms also are much more pronounced. If left to run its course, the condition is not rarely aggravated by the concurrence of nephritis and pyelonephritis (*q. v.*) which may lead to fatal termination.

Treatment.—As it is not always possible in the beginning to foresee the eventual course of the disease, and as the tendency even of mild cases toward chronicity is great, it is essential not to trifle with the affection, but promptly to employ all such therapeutic measures as will insure its early arrest and ultimate cure. The patient should be put to bed and on a mild diet (milk and Vichy water, milk gruel, fermented milk, and well-boiled vegetables). All spices, alcoholic beverages, coffee, and tea should be prohibited. To relieve pain, hyoscyamus is the remedy *par excellence*. It may be combined with citrate or acetate of potash and small doses (3 grains every four hours) of hexamethylenamine. Warm Priessnitz compresses are also of value. Where the pain persists, a suppository of codeine and extract of belladonna will be found to act well. With subsidence of the acute symptoms—usually after a week or two—it is advisable to begin to irrigate the bladder (under the most careful aseptic precautions) with a warm solution of boracic acid (5 i to 0 i) or of nitrate of silver or potassium permanganate (1/2000 or 1/1000). From ½ pint to 1 quart of the solution may be used for each treatment, and the irrigation may be repeated once a day or every other day. In mild cases boric acid solution (1 dram to 1 quart) alone may suffice.

Under this method of treatment the majority of cases of cystitis will recover in from four to eight weeks; provided, of course, the primary cause can be detected and removed.

Transition of simple acute cystitis into chronic is by far less common in children than in adults. The possibility of the disease being tubercular in nature, however, should always be borne in mind. (See p. 457.) For diagnostic purposes F. Hinman (Am. Jour. Dis. Child., May, 1919) strongly advocates cystoscopy and ureteral catheterization in all chronic cases, irrespective of sex or age of the child.

℞	Potassii Acetatis	3 i	4.00
	Ext. Hyoscyami Fl.	m. xvi	1.00
	Ext. Tritici Repens Fl.	3 i	4.00
	Inf. Uvæ Ursi	q. s. ad ℥ ii	60.00
	M.		

S.—One teaspoonful in water every four hours, for a child five years old. (Simple cystitis.)

In subacute or chronic colicystitis or pyelocystitis, urotropin is the remedy of choice. It acts best in combination with potassium citrate, 5 grains of each every 4 hours. In refractory cases we may have to

resort to frequent irrigations of the bladder and even of the ureters. Chronic cases will often yield to these procedures alone or in conjunction with biweekly hypodermic injections of autogenous vaccine. (See "Pyelitis".)

Vesical Calculi

(Stones in the Bladder)

Bladder stones sooner or later give rise to the following characteristic symptom complex: Vesical and often rectal tenesmus, strangury, partial or complete retention or incontinence of urine, difference in the force of the stream of urine with change in posture of the patient, and, after a protracted course, the usual symptoms of cystitis (*q. v.*). The urine may reveal the presence of either phosphate stones (phosphate and carbonate of lime, magnesia), oxalate stones (oxalate of lime), or urate stones (uric acid). Small concrements may escape with the urine; large ones, however, are apt to become impacted in the urinary canal and cause intense pain and grave nervous symptoms, *e. g.*, convulsions.

The diagnosis is based upon the aforementioned manifestations, upon feeling the stone in the bladder by rectal digital examination or by a sound introduced into the bladder, and upon an x-ray examination.

The development of stones may frequently be prevented by a bland diet (no meats), ample supply of water, and attention to the bowels. In cases of long standing operative interference is indispensable. Painful symptoms are relieved by means of hyoscyamus, or opium and belladonna suppositories.

Spasmus Vesicæ, Dysuria, Ischuria

(Anuria)

These conditions are etiologically correlated. In the majority of instances they are the result of vesical calculi, blood clots obstructing the urinary flow, phimosis, paraphimosis, vulvitis and vaginitis, cystitis, uric acid infarcts (in the newborn), sudden chilling of and injury to the lower portion of the abdomen, nerve affections (functional or organic), and priapism (in the male).

Treatment.—The treatment varies with the original cause. An attack is usually relieved by a hot bath, a suppository of codeine and extract of belladonna, and the administration of diuretics, such as sweet spirits of niter and extract. triticum repens.

℞	Potassii Citratis	℥ i	4.00
	Ext. Hyoscyami Fl.	m. xvi	1.00
	Ext. Tritici Repens Fl.	℥ i	4.00
	Syr. Simplicis	℥ iv	15.00
	Aq. Anisi	q. s. ad ℥ ii	60.00
	M.		

S.—One teaspoonful in water every three hours, for a child three years old.

Enuresis

(Bed-wetting. Incontinence of Urine.)

It is customary to distinguish two varieties of enuresis in children: enuresis diurna and enuresis nocturna. The first variety is but rarely met with in children, capable of differentiating right from wrong, excepting in those who willfully "wet" themselves, or in congenital deficiencies (spina bifida, q. v.). The second variety, on the other hand, occurs in a very great number of children, regardless of age, sex, intelligence or social conditions. The child may wet the bed one or more times every night, or at intervals of days or weeks; in the last event, it is usually due to willfulness, excessive drinking, or faulty diet. An inherited tendency and neurotic disposition seem to play an important part in the causation of enuresis, although the latter may exist independently of either of these causes in children apparently perfectly healthy.

The causes of enuresis may conveniently be arranged in two classes:

1. Functional.—The cases due to functional causes are purely neurotic in character. The urine is voided involuntarily either owing to *atony* of the sphincter vesicæ, or to a *spasmodic* condition of the detrusors vesicæ. In both cases there is a functional disturbance in the nervous apparatus of the urinary system. It is usually found that enuresis due to *atony* is associated with general debility, and often follows a protracted course of an exhausting disease. On the other hand, enuresis due to "spasm" is usually found in children who are irritable, who present an increased patellar reflex, are easily frightened, are subject to pavor nocturnus and similar nervous conditions.

2. Organic.—A great many cases arise from organic troubles. The child may suffer from organic disease of the spinal cord (spina bifida); cystitis; phimosis or paraphimosis (in the male); hypertrophy of the clitoris or adhesion of the prepuce (in the female); masturbation; undescended testicle; hernia, worms; vesical and renal calculi; tumors in the bladder; excessive quantity of lithiates or phosphates; constipation and accumulation of feces in the rectum; epi- or hypospadias; fissura ani; vulvovaginitis; diabetes, gonorrhea, simple or gonorrheal proctitis. Finally it may here be mentioned that hyper-

trophied tonsils and adenoids may be responsible for intractable enu-
resis.

Treatment.—In the treatment of enuresis it is of greatest moment
to systematically examine the patients for the organic diseases just
enumerated and to endeavor to eliminate every symptom suspicious
of organic disease. In absence of organic causes there is evidently a
neurotic case to be dealt with and the treatment must be adopted ac-
cordingly. The patient if old enough should be instructed not to ab-
stain from micturition when called upon by nature to do so, and small
children should be trained to void urine every three hours, and not be
permitted to withhold the urine for a longer period. This is very im-
portant, for it is often overdistention of, and decomposition of the
urine in, the bladder that prove the primary cause of the subsequent
secondary etiologic factors, (atony or hyperesthesia of the bladder,
presence of concretions, cystitis, etc.). It is also advisable to en-
courage drinking of water in cases of enuresis due to concretions,
cystitis, or gonorrhea, but to forbid it in other cases. The patient
is not to be permitted to sleep on his back, and it is often of advantage
to raise the foot of the bed in such a manner that the child's trunk
and head lie deeper than the pelvis.

In enuresis due to *atony* a general constructive treatment is indi-
cated. Plenty of good nourishment, change of air, cold spinal douches,
medicinal tonics and electricity are usually effective in bringing about
a cure. A moderate galvanic current is usually best; one pole is applied
to the symphysis or sacrum, the other to the perineum. The following
mixture is often very serviceable:

> ℞ Ext. Ergotæ Fl.   ℨ iii | 12.00
>   Ext. Rhus Toxicondron   ℨ i | 4.00
>   M.
>   S.—Five to 10 drops every four to six hours,
>   for a child six years old.

In incontinence of urine associated with hyperesthesia of the collum
vesicæ or *spasm* of the detrusors, antispasmodic treatment is indi-
cated, consisting of hot sitz-baths, avoidance of irritating food or drinks
and the administration of either extract of belladonna or hyoscyamus.
I usually prescribe the following:

> ℞ Ext. Hyoscyami Fl.   ℨ ss | 2.00
>   Natrii Bromidi   ℨ i | 4.00
>   Syr. Simplicis   ℥ i | 30.00
>   Aq. Anisi   q. s. ad ℥ ii | 60.00
>   M.
>   S.—One teaspoonful in water, three times a
>   day, for a child eight years old.

Counterirritation by means of sinapisms over the lumbosacral region often does well, and if everything fails, this class of cases is occasionally cured by gradual dilatation of the posterior urethral canal.

More recently some benefit has been claimed from the administration of pituitrin, in 5 drop doses (placed under the tongue or hypodermically) three times a day.

As to the treatment of enuresis from organic causes, nothing more will be said here than that each case must be treated as an individual disease in accordance with its etiology.

Remonstrance, severity and moral suasion will often cure cases of enuresis of nervous origin or those which continue from mere habit long after removal of the original cause.

Vulvovaginitis

(Cervicitis)

Clinically vulvovaginitis may be classified as follows:

1. *Catarrhal vulvovaginitis,* which is generally due to (*a*) lack of cleanliness or (*b*) chemical irritation.

2. *Traumatic vulvovaginitis,* which is due to (*a*) masturbation (?), (*b*) mechanical injury, or (*c*) indecent violence.

3. *Parasitic vulvovaginitis,* which is due to (*a*) oxyurides, (*b*) saprophytes, or (*c*) pathogenic bacteria, especially the gonococcus.

The first variety of vulvovaginitis is usually met in poorly nourished children of overcrowded tenement districts, who receive a thorough cleansing on very special occasions only. As a rule, these cases begin with vulvitis, the vagina becoming gradually involved by extension of the inflammation. Catarrhal vulvovaginitis is not always limited to the very poor, and the physician need not hesitate to suspect dirt even under the most elaborate apparel.

This variety of vulvovaginitis is also frequently observed in children whose genitalia are exposed to excessive wetting by irritating, decomposing secretions, and excretions—sweat, diarrheal stools, hyperacid urine—and to undue pressure and friction. In former years, when bicycle riding was a national fad, vulvovaginitis was not rarely met with in assiduous bicycle riders, undoubtedly as a result of the aforesaid causes. To the catarrhal type belongs also the vaginitis occasionally observed in the newborn.

The consideration of the second, traumatic, variety of vulvovaginitis does not, strictly speaking, belong to the domain of medicine, except as regards the treatment. We are dealing here with faulty habits and criminal traits which deserve serious attention on the part

of teachers, the clergy, and jurists. However, as it is the physician who is usually consulted first, a few points of information will prove useful to him, particularly as a warning not to be too hasty in expressing a positive opinion.

I believe that entirely too much stress is being laid by some authors upon masturbation as an etiologic factor of vulvovaginitis. It is much more probable that masturbation is a result rather than a cause of it, the undoubtedly existing irritated state of the erectile tissue inducing that bad habit.

The presence of foreign bodies in the vagina is not infrequently found to be the cause of vulvovaginitis. While some girls will introduce foreign bodies in the vagina with lascivious intent, the great majority of foreign bodies, (e. g., safety pins), will find their way in the vaginal canal accidentally, and should always be looked for, particularly in cases of long standing.

Occasionally cases of vulvovaginitis are encountered which are the result of indecent violence. The purulent discharge is either non-gonorrheal or gonorrheal, the latter only if the criminal who attempted rape had at the time been suffering from gonorrhea. It is well to remember that not every case of vulvovaginitis reported as due to rape, is really such, and unless the vaginitis is associated with actual penetration of the hymen and concomitant signs of inflammation due to violence, the physician should be very cautious in venturing a positive opinion.

Saprophytic microorganisms are responsible for a great number of cases of vaginitis. To them is attributable the vaginitis not infrequently met with after acute exanthematous diseases (e. g., measles and scarlet fever) and in conjunction with divers forms of cutaneous eruptions. The same cause accounts also for the vaginitis observed in strumous and debilitated children suffering from purulent discharges from the nose, ears, etc.—the discharges being carried to the vagina. Indeed, the number of cases of saprophytic vulvovaginitis would by far exceed all those arising from all other sources collectively were it not for the antagonistic action of the bacillus of Döderlein which normally inhabits the vagina. This vagina bacillus, which is anaërobic and may be cultivated on ordinary media, produces lactic acid during its growth, a quality to which is due the presence of lactic acid in the healthy vagina. In its presence saprophytes, as well as numerous other bacteria, such as the staphylococcus and streptococcus, are unable to develop, and within a short time perish. Gonococci, however, do not yield as promptly to the destructive effect of the vagina bacillus; hence, the frequency with which gonorrheal

vulvovaginitis is met, notwithstanding the resistance offered to the entrance of gonococci into the vagina by the stratified squamous epithelium lining it.

As stated before, contamination of the vagina by criminal assault is comparatively very rare. Much more frequently, infection takes place by voluntary sexual act or accidentally. Little girls sleeping with their parents, elder brothers, sisters, or nurses suffering from gonorrhea, may contract the disease by coming in contact with soiled bed clothes, cotton pads, or other articles used for cleansing purposes.

Gonorrheal vulvovaginitis runs a more or less virulent course, and in hospitals and asylums where many children are congregated in comparatively close quarters, and frequently make common use of infected bath tubs, toilets, etc., the disease is very apt to become epidemic as well as endemic. In one epidemic under my care in an orphan asylum, comprising over 100 cases, it required many months of very active treatment to eradicate the affection. Arrest of further spread of the gonorrhea was not effected until every patient was isolated and kept in bed for several weeks. A biweekly examination of every female inmate of the institution (including the nurses in charge) for vaginal discharge was continued for several weeks after disappearance of the last case of vaginitis.

Such procedures form the main prophylactic measures against the disease. Of course, the patients must be restricted from the common use of chambers, bedding, bath tubs, etc. In hospitals and asylums, admitting physicians should be particularly careful to exclude all children having a purulent vaginal discharge, unless provision be made for the isolation and treatment of such cases. This point is well worthy of consideration, since it would greatly aid in checking further transportation of the disaese. As the majority of cases of vulvovaginitis are observed among school children, a suggestion to the health authorities is, perhaps, in order, namely, to instruct the school inspectors to pay more attention to the detection and isolation of the cases of gonorrhea in children than they usually do.

Like gonorrhea in adults, that of children presents a marked tendency toward grave complications. Among 148 cases under my care, the following serious complications were observed: purulent ophthalmia, 7; local peritonitis, 4; proctitis, 3; arthritis, 4; adenitis, 12. Several cases of cervicitis,* endometritis and pyosalpinx; endocarditis, and pleuritis are on record. However, the more familiar one

---

*Hess reports that in 4 infants suffering from vaginitis he found (postmortem) an inflammation of the cervix with round-cell infiltration of its submucous tissue, and concludes that the average gonococcus infection involves the cervix rather than the vagina ("Gynoplastic Technology," by Dr. A. Sturmdorf, 1919).

becomes with the course of the disease and the best means of check-
ing and eradicating it, the less numerous will become the complica-
tions and sequelæ in his new cases.

After extensive experimenting I found that gonorrheal oph-
thalmia can best be prevented by frequent cleansing of the geni-
talia and hands of the patients, and by the employment of a large,
tightly fitting vulvar pad. The latter should be changed for a clean
one at least every three hours. The child should wear one-piece night
drawers during the night as well as during the day. The ophthalmia may
sometimes be arrested in its incipiency—I succeeded in two cases—by in-
stillation of silver solutions after Credé's method. In view of the
unusually rapid progress of the ophthalmia, unfortunately, it is not
often that the physician has the opportunity to resort to the pro-
phylactic measures, and nothing else remains but to treat the disease
actively and skillfully (see "Ophthalmia Neonatorum"), and, if not
already involved, to endeavor to save the other eye from the dreadful
infection.

Involvement of the cervix and fundus of the uterus and adnexa,
secondarily to gonorrheal vulvovaginitis, results in most instances
from injudicious use of douches by forcing the vaginal discharge up-
ward into the uterus, Fallopian tubes, etc. The treatment therefore
should not be intrusted to the inexperienced.

Many years ago I called attention (Amer. Medico-Surg. Bull., May
30, 1896) to the occurrence of gonorrheal proctitis as a complication
of vulvovaginitis. The rarity with which this complication is ob-
served, notwithstanding the constant exposure of the anus to the
gonorrheal vaginal discharge, would seem to prove the comparative
immunity of the skin and mucous membrane of the anus and rectum
to gonorrheal infection. Moreover, proctitis usually does not develop
until late in the course of the vaginitis, i. e., until the skin of the anus
and the adjacent structures have become abraded and denuded by the
continued irritation of the vaginal discharge, or by scratching for the
relief of the not infrequently accompanying intense itching.

The diagnosis of gonorrheal proctitis is rendered positive by the
presence of the gonococcus in the mucopurulent stools.

Like the former complication, arthritis, the so-called gonorrheal
rheumatism, also develops late in the course of vulvovaginitis. In the
majority of cases the inflammation is limited to one joint, usually that
of the knee, and occasionally ends in suppuration and ankylosis.

Inguinal adenitis is quite a frequent complication. The glandular
enlargement may increase up to a well-marked bubo. It sometimes
suppurates as a result of an additional infection by pus microbes.

The differential diagnosis between the different varieties of vulvovaginitis can readily be made by bearing in mind the previously mentioned classification. No examination should be considered complete without a very careful microscopical scrutiny of the vaginal discharge. In doubtful cases a culture will settle the diagnosis. Furthermore, it is well to remember that several etiologic factors may be operative in the production of the vaginitis in one and the same patient. Hence, the finding of pinworms, for example, in the vagina should not lead us to conclude the absence of gonococci.

The *active treatment* of vulvovaginitis varies greatly with the cause. Nongonorrheal cases usually yield promptly to removal of the etiologic factors (*e. g.*, foreign bodies) and to cleansing of the genitalia with salt, boric acid, or 2 or 3 per cent permanganate of potash or sulphocarbolate of zinc solutions and daily sea salt sitz-baths. In some cases insufflations of tannic acid, in powder form, act very beneficially. Gonorrheal vulvovaginitis should be treated by instillation into the vagina (through a soft-rubber catheter) once a day or every other day of ½ ounce of a 2 per cent to 5 per cent solution of nitrate of silver, followed by neutralization with salt water; or a 10 per cent solution of silvol, argyrol, or solargentum. After subsidence of the active symptoms douches with mild antiseptics will suffice. The suggestion recently made by a learned clinician to incise and dilate the hymen (in order to allow free application of antiseptics to the vaginal wall) is here mentioned only to be strongly condemned.

Gonorrheal urethritis in male children is treated the same as in the adult.

It is well to remember that recurrence of the affection after a period of latency is frequent even under the most careful method of treatment. No case of gonorrheal vulvovaginitis, therefore, should be considered cured unless three or more thorough microscopic examinations of the vaginal discharge prove the absence of gonococci and pus.

Masturbation

(Onanism, Thigh Friction)

Production of venereal orgasm by hand, or other unnatural means, is a very common vice among school children, who usually acquire the vicious habit from older playmates, or erotic governesses, etc. Masturbation is quite common among mental defectives.

Occasionally masturbation is observed in younger children and even in infants. The latter may be seen to rub their thighs against each other or against the bosom of the nurse, or to exert peculiar

rocking motions and fall back in a more or less marked state of exhaustion.

The effects of masturbation vary with the frequency and duration of the habit and the physical condition of the child. In the majority of cases masturbation produces physical and mental debility, especially depression of spirit, headache, palpitation of the heart and emaciation. In boys we may suspect masturbation by excessive elongation of the penis, in girls by the presence of vulvitis, and often stretching of the hymen. Boys are apt to suffer from nocturnal seminal emission and later also from impotence.

In remedying this evil, it is essential to remove all local sources of irritation, such as phimosis, hypertrophy of the clitoris, pinworms, etc. Infants should be restrained from practicing the bad habit by mechanical devices (separation of the thighs, tying of the hands). Older children should be placed under proper surveillance and in suitable spiritual surroundings (change of school or nurses!). The general health should be improved by outdoor exercise, cold shower baths, and by nutritious but bland diet (no liquors). Bromides are indicated to subdue sexual excitement. Dime novels should be eliminated from the child's reading room.

Menstruatio Precox

Genuine precocious menstruation in early childhood is of very rare occurrence. If it does occur, it is usually associated with general bodily and mental overdevelopment, most probably due to pituitary overactivity (see p. 571). The diagnosis of menstruatio precox should not be made until vaginal bleeding from local injury, from papillomatous growths, prolapse of the urethral mucous membrane, and hemophilia have been excluded.

Precocious menstruation, being free from serious consequences to the general health, calls for no therapeutic measures, except perfect rest during menstruation.

Gangrene of the Genitalia

(DIPHTHERIA VULVÆ, NOMA VULVÆ)

Gangrene of the genitalia (vulva, penis, scrotum, etc.) usually develops secondarily to grave local inflammatory processes in the vicinity. More rarely it is primary in nature (after too liberal use of strong antiseptic dressings in open wounds, e. g., carbolic acid gangrene in circumcision; the result of direct violence, e. g., stuprum), or

occurs in connection with diphtheria, dysentery, typhoid, and similar affections.

Whatever the cause, the prognosis is always very serious, fatal termination usually taking place within about ten days from the onset, unless we succeed in checking the spread of the gangrene by early cauterization or excision of the affected part. Diphtheria antitoxin is deserving of trial even if a smear or culture of the gangrenous deposit proves negative.

CHAPTER XII

DISEASES OF THE NERVE SYSTEM*

A. Organic Diseases

Hydrocephalus, Congenital and Acquired

(Acute and Chronic Dropsy of the Brain)

Hydrocephalus is an accumulation of serous, slightly albuminous fluid within the cranium. It may be of prenatal origin or develop during or immediately after birth as a result of traumatism to the

Fig. 169.—Congenital hydrocephalus. (Dr. M. Knowlton.)

head; or it may make its appearance at any other time during infancy and childhood, either as a primary affection or secondarily to a number of acute and chronic diseases. The fluid may collect in the subdural space (*external hydrocephalus*) or in the ventricles (*internal hydrocephalus*). The hydrocephalus may run an *acute* or *chronic*

*For "Congenital Malformations," see p. 174.

Fig. 170.—Congenital hydrocephalus with spina bifida. Every few weeks the hernial sac would fill up with cerebrospinal fluid and rupture. A few days before its occurrence, there were distinct symptoms of brain pressure, including convulsions.

Fig. 171.—Same case as Fig. 170 showing distended spina bifida before escape of the spinal fluid.

course. For detailed description of *chronic*, more particularly, congenital hydrocephalus, the reader is referred to the chapter on "Hydrocephalic Amentia," p. 710.

In order to obtain a clearer understanding of acute hydrocephalus, it is best to study its symptom complex in connection with the affections which form its underlying pathologic basis, as follows:

1. *Meningitis Serosa (Acute Internal Hydrocephalus—Quincke)*.— This condition may complicate acute febrile diseases, such as pertussis, influenza, pneumonia or typhoid, or set in primarily in the same manner as serous effusions in other cavities of the body, *e. g.*, pleuritis, pericarditis, and the like; undoubtedly also as a result of bacterial

Fig. 172.—Hydrocephalus following meningitis.

invasion or traumatism. The quantity of fluid varies, and upon its amount and the pressure it exerts upon the surrounding structures depends the clinical course. If the pressure is great, we have sopor, spasms, strabismus and nystagmus, and the head may assume an enormous size. In infants, the fontanelles are bulging, the cranial sutures are separated, and the frontal bone protrudes markedly forward. In the early stages of serous meningitis there is moderate

temperature.   If recovery does not take place within a reasonable
time the patient usually succumbs to gradual emaciation and increas-
ing cachexia.

2. *Tuberculosis of the Meninges or Brain. (Acute Internal Hydro-
cephalus)*.—The hydrocephalus usually develops slowly. The infant may
be affected with recurrent attacks of diarrhea, occasional vomiting, low
fever, apathy, weakness of the extremities, and spells of sudden,
piercing outcries, especially during the night. Older children often
complain of severe headache, are languid and refuse to participate in
the plays of their comrades. Gradually the symptoms grow worse.
Vomiting, rigidity of the neck, and paralysis of the cranial nerves
make their appearance, and, in a short time thereafter, the typical
symptoms of cerebrospinal meningitis set in, which sooner or later
lead to fatal termination. Ordinarily the hydrocephalus is not as
marked as in meningitis serosa.

3. *Wasting Diseases, Acute and Chronic*.—The hydrocephalus is
usually slight, and hence does little or no damage to the brain. This

Fig. 173.—Acquired acute hydrocephalus, following acute gastroenteritis and com-
plicating rachitis. Note peculiar arching of forehead.

is true especially of hydrocephalus accompanying rachitis, and acute
and chronic gastroenteritis. With subsidence of the underlying cause
the cranial distention generally disappears; the disfigurement of the

skull, however, may remain permanent. This form of hydrocephalus is sometimes spoken of as spurious.

The symptomatology of chronic hydrocephalus, which occasionally follows serous meningitis is essentially identical with that of congenital hydrocephalus, except that the tendency to idiocy is not as great; indeed some of the children grow up with practically normal mentality. Herein, of course, are not included the cases of hydrocephalus associated with cerebral tumors.

The occasional concurrence of dyspituitarism and hydrocephalus has been emphasized by Harvey Cushing,[1] L. J. Pollock,[2] Fröhlich,[3] Neurath,[4] Strauch,[5] by the author,[6] and others to whose instructive papers on the subject the reader is referred for further knowledge.

Treatment.—The treatment varies with the etiologic basis of the affection. Lumbar puncture is always useful whenever pressure symptoms become evident, and in young infants we may also resort to puncture of the lateral ventricles. As hydrocephalus is sometimes a manifestation of congenital syphilis, specific treatment is worth trying, even though the Wassermann reaction may prove negative. For further information the reader is referred to the chapter on "Idiocy".)

Anemia of the Brain

(HYDROCEPHALOID)

This condition is usually the result of excessive loss of body fluids (repeated hemorrhages), general grave anemia, exhaustion from acute (rarely chronic), gastrointestinal diseases, interference with the blood supply of the brain (pressure on the part of tumors), etc. If the anemia is moderate, it is manifested principally by syncope.

Anemia of the brain occuring in violent gastroenteric affections (with profuse vomiting and diarrhea) is generally spoken of as "hydrocephaloid," so designated by Marshall Hall, who first described the symptom complex. Hydrocephaloid is characterized by a stage of *excitation*: flushed face, fever, restlessness, jactitations; and one of *prostration*: pallor, sunken face, irregular pulse and respiration, cold extremities, subnormal temperature, sunken fontanelles, stupor with half-closed eyes, hazy corneæ, coma, convulsions, and, as a rule, death.

---

[1]"The Pituitary Body and its Disorders," 1911.
[2]Hypopituitarism in Chronic Hydrocephalus, Jour. A. M. A , Jan. 30, 1915.
[3]Wien. klin. Rundschau, No. 45, 1901.
[4]Wien. klin. Wchnschr., No. 2, 1911.
[5]Jour. A. M. A., June 14, 1919.
[6]"The Backward Baby," p. 56.

Occasionally hydrocephaloid yields to energetic treatment, which consists of external heat, transfusion, stimulation by entero- and hypodermoclysis, intravenous saline, sterile camphorated oil and strychnine hypodermically; champagne and small quantities of food by mouth. Fresh air.

The brain of infants dying from cerebral anemia is pale, watery and softer than normal.

Hyperemia of the Brain

The hyperemia may be active or arterial; passive or venous.

Active hyperemia may occur as a result of sunstroke, traumatism, mental or physical overexertion, overstimulation by exhilarating beverages or drugs, hysteria, onset of acute infectious diseases, etc.

It is manifested by deep redness of the face, congestion of the conjunctivæ, contraction of the pupils, hot skin, high temperature, accelerated pulse, strong pulsation of the carotids and temporals, ringing in the ears, intense headache, excessive thirst, and in severe cases convulsions, dilirium, distention of the fontanelles, and other symptoms of meningeal irritation.

Passive hyperemia of the brain is caused by passive congestion of the cerebral veins owing to cardiac debility, grave pulmonary affections (edema, pertussis, etc.), compression of the veins in the neck, etc.

The symptoms of passive hyperemia are those of exhaustion, apathy, somnolence, cyanosis of the face and dyspnea.

Treatment.—The treatment depends upon the original condition. It is more or less symptomatic—antiphlogosis in the active, stimulation in the passive variety of hyperemia.

Upon the underlying cause also depends the final outcome. Protracted hyperemia sooner or later leads to meningitis, rupture of cerebral blood vessels, and dropsical effusion in the cranial cavities.

General Remarks on Cerebral or Central Paralysis and Brain Localization

"Cerebral paralysis," so-called, is not an independent brain disease, but merely a symptom occurring in connection with a number of congenital and acquired brain affections. Depending upon the extent of the lesion in the brain the paralysis may appear either in the form of hemiplegia, double hemiplegia, or monoplegia.

Hemiplegia is the result of a lesion (disease or trauma) in one cerebral hemisphere. The paralysis is situated on the side opposite that

BRAIN LOCALIZATION

SEAT OF LESION	USUAL MANIFESTATIONS AND THEIR SEAT
Central convolutions:	
1. Upper third.	Paralysis of leg, opposite side; convulsions.
2. Middle third.	Paralysis of arm, opposite side; convulsions.
3. Lower third.	
(a) Upper part.	Paralysis of the muscles of one-half of the face.
(b) Lower part.	Paralysis of the muscles of the lips and tongue.
Frontal convolutions.	Disturbance of speech.
Parietal convolutions.	Disturbance of cutaneous and muscular sensibility.
Occipital convolutions (especially cuneus).	Hemiopia; loss of visual memory.
Temporal convolutions.	Disturbance of hearing, opposite side, and sense of smell.
Centrum ovale.	Monoplegia, hemiplegia, hemiopia, word deafness and aphasia; convulsions.
Central ganglia (caudate and lenticular nuclei).	Hemiplegia and hemianesthesia.
Optic thalamus.	Disturbance of vision up to blindness.
Internal capsule.	Hemiplegia and hemianesthesia, and sometimes loss of special senses.
Corpora quadrigemina (anterior pair).	Oculomotor paralysis, reeling gait, possibly total blindness and deafness.
Crura cerebri.	Hemiplegia with cross paralysis of oculomotor nerve.
Pons and medulla (one-half).	Hemiplegia with cross paralysis of facial nerve; hemianesthesia; also involvement of other cranial nerves, e. g., hypoglossal, abducens, varying with the height of the lesion.
Cerebellum.	Ataxia, vertigo, and vomiting.

of the lesion. Motile power may be completely abolished or only partially so (paresis). Sensation may remain intact, but is lost if the brain lesion is in the internal capsule and extends to the sensory fibers. The paralysis is associated with spastic rigidity of the affected muscles, exaggeration of the deep reflexes, implication of some of the cranial nerves, such as the facial (palsy of the lower part of the face), hypoglossal (deviation of the tip of the tongue to healthy side), and ocular nerves (nystagmus, hemianopsia, and optic atrophy), and occasionally, in a left-sided lesion, also with motor aphasia. As the paralysis becomes chronic the paretic musculature shows a tendency

to arrest of development, tremor and athetosis; and epilepsy and mental impairment up to total idiocy make their gradual appearance.

Diplegia (double hemiplegia) may be the result of two separate attacks of hemiplegia. More frequently it develops with one attack as a sequel of extensive brain lesions in both cerebral hemispheres or in the pons and medulla (affecting both lateral halves). If only one side of the pons is involved, we have cross paralysis of the extremities on one side and of the facial nerve on the other side.

In double hemiplegia, in addition to the symptoms enumerated under hemiplegia, functions may suffer which escape ordinary hemiplegia, *e. g.*, that of swallowing and, perhaps, that of micturition. Occasionally it is accompanied also by paralysis of the tongue, giving rise to symptoms which closely resemble those associated with bulbar paralysis. However, there is no wasting of the tongue, nor change in the electric reaction; hence, is spoken of as "pseudobulbar paralysis."

Monoplegia as a primary manifestation of a cerebral paralysis is rare. More frequently it is met in the regressive stage of the aforementioned two types of paralysis or in connection with lesions of the spinal cord or peripheral nerves. Cerebral monoplegia usually arises from a limited lesion in or near the cortex (*e. g.*, in simple encephalitis), less frequently from smaller capsular lesions involving individual nerve bundles for the face, arm, leg, etc.

Intracranial Hemorrhage

(Meningeal Hemorrhage, Hemorrhage in the Brain)

We had occasion (p. 208) to direct attention to hemorrhages resulting from obstetrical injuries. This space will be devoted to the discussion of intracranial hemorrhages occuring during infancy and childhood. The usual sites for intracranial hemorrhages are as follows: neighborhood of the large central ganglia, pons, meninges, convolutions, cerebellum, crura cerebri or medulla.

They may occur as a result of trauma, such as a blow or fall upon the head; in association with meningitis, infectious diseases, purpura, pertussis (as a result of severe venous congestion); sinus thrombosis, syphilis (syphilitic arteritis), richly vascular tumors; nephritis and hypertrophy of the heart (owing to increased blood-pressure), etc.

In the majority of instances the symptomatology is at first indefinite and inseparable from that of the fundamental disease. Where the hemorrhage is extensive, the symptom complex resembles in its entirety that observed in intracranial hemorrhage in adults. Thus:

unconsciousness, convulsions, slow, irregular breathing, slow and full pulse, coma and death, or partial recovery with persistent focal signs, especially paralysis (hemiplegia, diplegia or monoplegia).

Treatment.—The treatment consists of an ice cap to the head, counterirritation (wet cups to nape of neck), perfect rest, light nutritious diet, and later, ergot and the iodides. In traumatic cerebral hemorrhage early operative interference is indicated. Pressure symptoms may be relieved by lumbar puncture.

Embolism of the Brain Arteries

Cerebral embolism like hemorrhage is rarely observed in children. It is occasionally met in connection with severe valvular heart disease, and acute infectious and pyemic processes, and most frequently affects the arteria fossae Sylvii.

The symptomatology of embolism is practically the same as in cerebral hemorrhage (*q. v.*), except that in the former the signs of cerebral compression and shock are not as persistent and as severe. Furthermore, the existence of valvular heart trouble decides in favor of embolism. The onset is usually sudden (occasionally preceded by headache, vomiting, etc.), with convulsions, coma, etc., followed either by early death, or partial recovery, with remaining focal symptoms, especially hemiplegia and aphasia. In septic embolism there is irregular fever.

Treatment.—The treatment is the same as in cerebral hemorrhage. Antisyphilitic treatment may be tried in cases of doubtful origin.

Sinus Thrombosis

Thrombosis in the large sinuses of the dura mater is most frequently observed in debilitated infants. Two forms are distinguished: passive or marantic, being the result of retardation of the venous blood current in severe cardiac, gastrointestinal, or other exhausting diseases; active or infective, occurring in connection with inflammatory processes in the vicinity, *e. g.*, ear, nose, eyes, etc.

Passive sinus thrombosis is usually limited to the longitudinal sinus and is manifested by symptoms of exhaustion and collapse and those of hydrocephaloid plus local edema and distention of the veins of the head and face.

Active sinus thrombosis usually involves the transverse and petrosal sinuses and is characterized in addition to the aforementioned phenomena by more or less marked septic symptoms (vomiting, chills and fever, etc.), hemorrhagic infarcts and embolism, *e. g.*, in the lungs, spleen and other organs of the body.

The differential diagnosis between the two varieties of sinus thrombosis is quite difficult, but somewhat facilitated by lumbar puncture, which in the infective form reveals in the hemorrhagic cerebrospinal fluid numerous bacteria (strepto- or staphylo-, or pneumococci). When the longitudinal sinus is involved, there are epistaxis, cyanosis of the face, edema of the soft tissues of the frontal, parietal and temporal regions and frontal sweating. When the transverse and petrosal of one side are affected, corresponding collapse of the jugular vein and edema of the mastoid region result. When the cavernous sinus is implicated, exophthalmos, chemosis of the conjunctivæ and lids are the distinctive signs.

Treatment.—Where a diagnosis can be established early, opening of the sinus may prove a life-saving operation in septic sinus thrombosis. Otherwise little can be accomplished in the way of therapy. In marantic sinus thrombosis, active stimulation may act well in some cases. The prognosis, thus being so extremely grave, our attention should be directed principally toward prophylaxis, especially as regards extension of the suppurative process from neighboring structures.

MENINGITIS ACUTA

(MENINGITIS CEREBROSPINALIS)

Meningococcic, Pneumococcic, Tuberculous, Streptococcic, Etc., Meningitis[1]

Meningitis may be primary or secondary in nature. *Primary* meningitis may be the result of traumatism (may involve both the dura mater—pachymeningitis hemorrhagica—and pia mater, but usually the former) or may be due to direct infection of the meninges by the diplo-

[1]Our venturesome attempt to disrupt the time-worn mode of grouping of the different varieties of meningitis is based upon the following considerations. 1. The symptom complex of fully established meningeal inflammation is practically identical in all forms of the disease, and differs only in the degree of mildness or severity of the attack, which depends upon the extent of the lesion, the susceptibility and the power of resistance of the patient to the microbic toxin and its baneful effects. 2. The same lack of distinction is observed in the pathologic anatomy of the divers forms of meningitis, except that in tuberculous meningitis we find in addition to the usual inflammatory process, local or general dissemination of tubercles, which, however, are not manifested by special clinical sumptoms. 3. Even the formerly accepted view as to the characteristic distribution of the inflammation in certain varieties of the affection, *e. g.,* the so-called "vertical" or "basilar" meningitis, etc., is no longer scientifically tenable in a strict sense of the word, since meningitis of the convexity of today may, by extension, become that of the base the day following and *vice versa*. With these considerations in view, and appreciating also the fact that a positive differential diagnosis of the variety of meningitis can be made only by the findings of the etiologic factors in the cerebrospinal fluid obtained by lumbar puncture, we feel fully justified to discard the subdivision of meningitis into "serous," "purulent," "epidemic," "posterior-basic," etc., and to classify the disease from an etiologic point of view. Just as we speak of "tuberculous meningitis," we speak also of meningococcic, pneumococcic, streptococcic, influenzal meningitis, etc.—a classification which is not only scientifically correct, but at once offers a clue as to the etiology, mode of treatment, and prognosis.

coccus intracellularis meningitidis* (Weichselbaum, Leichtenstern and Jäger) and other pathogenic bacteria, *e. g.,* streptococci or staphylococci, and affect the pia mater of the brain as well as the cord—cerebrospinal meningitis. *Secondary* meningitis is due to extension of the infection from neighboring or more remote parts. This form includes the tuberculous, or pneumococcus meningitis, as well as the meningitides which are met with in divers acute infectious diseases, such as influenza, typhoid fever, erysipelas, otitis, diphtheria and the like. The infection spreads either by continuity (throat, nose or ear), by the lymphatics, or by the blood vessels.†

Meningitis is a disease peculiar to early childhood, the majority of cases occurring in the first three years of life. It prevails principally, often in epidemic form (epidemic cerebrospinal meningococcus or malignant meningitis) during the late winter and spring months, at a time when, with rapid changes in the weather and crowding of the children in stuffy rooms, "colds" and their sequelæ are fiercely rampant. It is observed also sporadically during all seasons of the year. Delicate children are more prone to be attacked than robust ones, this being the case especially with tuberculous meningitis, which is frequently the culmination of latent tuberculosis of other organs of the body.

The mode of onset of the disease varies greatly. It is usually abrupt in primary meningitis, rarely preceded by a few indefinite signs of ill health, such as anorexia, restlessness and headache. In secondary meningitis the attack, as a rule, develops more insidiously and is often obscured by the symptomatology of the preceding affection. Meningitis supervening latent tuberculosis with few exceptions is particularly prone to be gradual in its development. In these cases the child may for weeks manifest apathy, anorexia, vomiting, wasting, occasional rise of temperature, and other symptoms corresponding to the seat of the original lesion (*e. g.,* caseation of the bronchial, mesenteric, or intestinal glands; bone or joint disease, etc.).

Acute meningitis, be it primary or secondary, gives rise to dizziness, headache, nausea, projectile and usually persistent vomiting, rise of temperature, jactitations up to convulsions, alternating with drowsiness, stiffness and pain in the neck. This group of symptoms, while *per se* not at all characteristic, is nevertheless strongly suspicious of the disease. Finding a patient in this condition we should at once carefully

---

*Type A, B, C, or D. See p. 78.

†By special care in preparation of mediums and other details, Marshall A. Barber. Captain, S. C., N. A., and J. F. Fleming, First Lieutenant, M. R. C., have obtained positive blood cultures in twelve cases. Recent experience would indicate that with early diagnosis and proper laboratory technic the meningococcus may be grown from the blood in from 50 to 80 per cent of all cases of epidemic meningococcus infection. W. W. Herrick: J. A. M. A., Aug. 24, 1918.

examine him for the following more or less pathognomonic physical signs and symptoms of meningitis:

Opisthotonos or Rigidity of the Neck and Brudzinski's Sign.—This symptom is elicited by placing the hand under the patients' occiput and flexing the head upon the chest. In meningitis the neck will be found stiff and painful. Forcible flexion of the head upon the chest usually produces synchronous flexion of the legs upon the abdomen (Brudzinski's sign). The child instinctively assumes a lateral position, as the dorsal position proves very painful by pressure of the head against the pillow. Rigidity of the neck is present at one time or another in all cases of meningitis. It is especially pronounced in cases in which the inflammation begins at the posterior part of the brain. As the disease advances the rigidity extends to the muscles of the back and extremities, gives rise to a spasmodic rigidity of the body in which the trunk is arched forward and the shoulders and buttocks are thrown backward while the legs, as a rule, are flexed upon the thighs—opisthot-
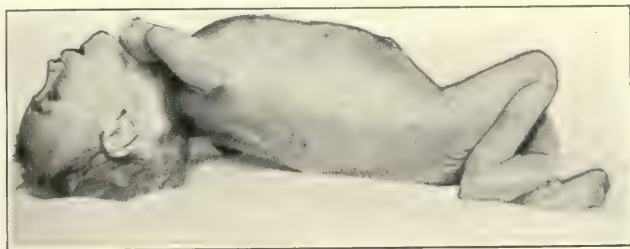
Fig. 174.—Epidemic cerebrospinal meningitis. (After Pfaundler and Schlossmann.)

onos. Occasionally the forearms are extended and the fingers clenched in the palm.

Kernig's Sign.—This symptom consists of inability of the examiner to extend the patient's legs with the thighs flexed on the abdomen. It is met in the majority of cases of meningitis, but it is not entirely pathognomonic of the disease, since it is observed also in other affections, *e. g.,* typhoid fever, and occasionally also in normal infants. In conjunction, however, with the other meningeal symptoms Kernig's sign is very helpful in the diagnosis.

Babinski's Reflex.—Irritation of the plantar surface of a patient suffering from meningitis produces extension of the great toe with flexion of the other toes. It is a characteristic sign of disease of the pyrimidal and lateral tracts of the cord, hence is more apt to be observed in very diffuse forms of inflammation (tuberculosis) of the meninges and underlying structures than in the localized forms of the

disease. This sign is least reliable in infants under two years of age, but is of corroborative value in older children.

Leichtenstern's Sign.—This consists of lightning-like contraction of the whole body on striking any part of the bony framework with the percussion hammer. It is a symptom of meningitis, principally during the stage of irritation.

Reflexes.—In the early stages of meningitis the skin and tendon reflexes are somewhat exaggerated, but with the gradual loss of muscular power they disappear partially or wholly.

Changes in the Eyes.—Intolerance to light and contraction of the pupils form early symptoms of meningitis. Dilatation or inequality of the pupils is usually met with later. The inequality is usually transient and variable, present at one time and absent at others: now one pupil, now the other may be the larger. Strabismus and nystagmus are observed in advanced stages of the affection. Examination of the fundus reveals, in the majority of cases of tuberculous meningitis, optic neuritis or papillitis, and tubercles in the choroid. Optic neuritis is occasionally found also in other varieties of meningitis, chiefly when the base is involved. After the first week the child often keeps the eyes open staring immovably into distance.

Vasomotor and Cutaneous Disturbances.—Cutaneous irritation is usually followed by a vivid and enduring congestion of the skin— *taches cérébrales* (Trousseau's sign). This symptom is not very significant, being observed also in other infectious diseases, *e. g.,* typhoid fever. Eruptions of the skin—erythema, herpes, urticaria and purpura— are quite frequent. Purpuric spots are especially common in fulminant cases (hence often spoken of as spotted brain fever). They vary in size and may coalesce to form dark diffuse extravasations into the skin.

McEwen's Sign.—With the patient in an upright position and his head inclined to one side, percussion over the junction of the lower portions of the frontal and parietal bones gives a tympanitic note. This situation corresponds to the anterior horn of the lateral ventricle, and the note is caused by the presence of fluid in the ventricle. Hence it is most frequently observed in the tuberculous variety of meningitis, where there is an accumulation of fluid in the ventricles. This sign is not pathognomonic before complete ossification of the skull.

Mental State.—In the beginning of the disease the children are usually very irritable. They twitch, grind the teeth, start up with a cry of alarm when disturbed, are annoyed by the least sound in the room; but as the meningitis progresses, or in the tubercu-

lous variety often at its very inception, the patient gradually enters into a state of apathy, stupor and coma.

Blood.—There is generally a high leucocytosis (as high as 45,000 to 55,000 per cubic millimeter, rarely below 20,000) in the nontuberculous forms.

The experienced clinician, in order to arrive at a conclusion, rarely needs to wait for the synchronous inauguration of all of the aforementioned symptoms. Indeed, it is quite uncommon to meet with cases which present such an array of typical phenomena. One seldom errs in the diagnosis where persistent vomiting, convulsions, rigidity, photophobia and stupor are grouped together. However, the mere diagnosis of meningitis is not sufficient. It is also the cause and variety we are interested in.

Cerebrospinal Fluid.—With the latest improvements in the technic of examination of the cerebrospinal fluid obtained by lumbar puncture, numerous doubtful points of diagnosis can be cleared up which before the introduction of this diagnostic procedure forever remained a mystery.

Normal cerebrospinal fluid is a clear neutral or slightly alkaline fluid, containing but a small proportion of salines, a small quantity (0.05 to 0.1 per cent) of serum globulin, a trace of cholin and a sugar-reducing agent (0.5 per cent). It is not spontaneously coagulable. Its specific gravity varies between 1,007 to 1,009.

In normal individuals it escapes through the puncture needle at a low pressure, usually drop by drop. The pressure may accurately be measured by the manometer, but the experienced eye can well appreciate the amount of tension by observing the force of the jet.

The *pressure* is usually increased in divers meningeal irritations and is particularly high in tuberculous and hydrocephalic conditions. As the stream may be altered by the position of the patient, by the viscosity of the fluid, by interference with the flow in its path etc., the semiologic importance of pressure is rather slight.

The *color* of the cerebrospinal fluid may be altered by accidental or pathologic admixture of blood, pus or pigment. In acute bacterial meningitis the discoloration varies from slight cloudiness to a well-defined purulent turbidity. In tuberculous miningitis the fluid is usually clear or slightly opalescent; on standing a thin film forms on the upper surface. The presence of blood is readily recognized and may be due to accidental admixture from the puncture wound or to hemorrhagic pachymeningitis.

The *bacteriologic* examination of the cerebrospinal fluid is of inestimable clinical value, since it often furnishes reliable information not only as to early diagnosis, but to the prognosis and treatment as well. Too much stress cannot be laid upon the fact that, in order to obtain conclusive pathologic data, the examination of the fluid should be intrusted to one thoroughly experienced in bacteriology and microscopy. Negative results in the majority of instances are due to skepticism and faulty technic. Occasionally repeated examinations are required. Nearly all kinds of microorganisms have been found. Careful search for the
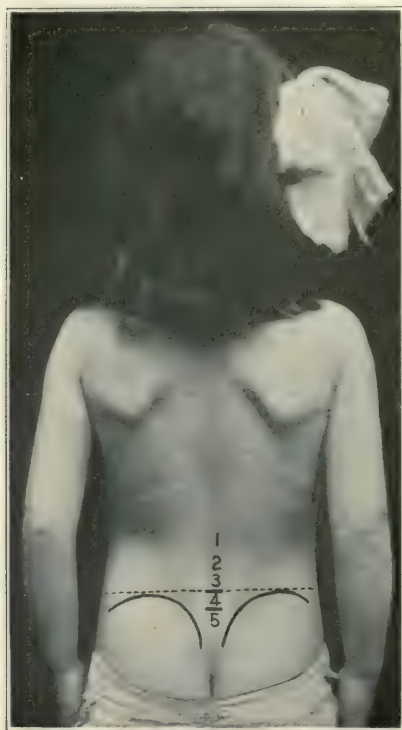
Fig. 175.—Lumbar puncture. The patient is put near the edge of a table in sitting or lying posture, with the vertebral column strongly arched forward. The puncture is made with a thin, hollow exploratory needle in the lumbar region, in the third or fourth intervertebral space, at a point corresponding to a line drawn between the superior crests of the ilia.

tubercle bacillus should be made in all cases of meningitis, regardless of clinical data. The finding of the tubercle bacillus in the cerebrospinal fluid at a glance settles the diagnosis, where volumes of descriptions of differential features at best fail. The same applies for the diplococcus intracellularis meningitidis, and other pathogenic bacteria.

CEREBROSPINAL FLUIDS

(After Dr. A. Sophian)

	Normal	Meningism	Poliomyelitis Polioenceph- alitis	Cerebrospinal Meningitis	Streptococcus, Pneumococcus, Influenza, etc., Meningitis	Tuberculous Meningitis
Color	Clear	Clear	Clear	Cloudy—pus sediment	Cloudy—pus sediment	Clear—white flakes—fibrin- network
Pressure	Low—escapes slowly drop by drop	+	+	+ +	+ +	+ + +
Quantity	Little—few c.c.	+ (up to 50 c.c. or more)	+ (up to 50 c.c. or more)	+ + (up to 100 c.c. or more)	+ + (up to 100 c.c. or more)	+++ (up to 100 c.c. or more)
Cytology	Few cells, leu- kocytes and endothelial	Few cellular elements	Cells increas- ed (+) in number. Lym- phocytes 90% or more	Cells numerous + + + (Polynu- clear up to 100%)	Cells numerous + + + (Polynu- clear up to 100%)	Cells numerous ++ (Lymph- ocytes up to 90%)
Bacteri- ology	Sterile	Sterile	Sterile	Meningococcus	Infecting or- ganism	Tubercle bacil- lus
Albumin (nitric acid test)	Faint trace	Trace	Trace	+ + +	+ + +	+
Fehling's Solution	Reduces	Reduces	Reduces	Unreliable	Unreliable	Unreliable
Globulin Test	Negative	Negative	Positive in early stages	+ + +	+ + +	+ +

For the detection of the microorganism we may use stained smears (the specimen having been obtained from the coagulum that forms in the fluid on standing or after centrifugation), cultures, or inoculation methods. Where rapid decision is demanded the last two procedures are not adoptable, but as their scientific accuracy is incontestable they are not rarely indispensable in cases of obscure origin and especially in mixed infections.

Cytodiagnosis.—This is based upon the histologic study and deter- mination of the number and nature of the formed elements in the cere brospinal fluid. Normally this fluid contains very few cells, so few that in a smear obtained from the deposit after centrifugation only two or three leucocytes may be visible in the microscopic field. The presence of leucocytes in great numbers constitutes anatomic evidence of a meningeal lesion—namely, of tuberculous nature, where lymphocytes[*] (mononuclears) prevail, and nontuberculous, where polymorphonuclear leucocytes predominate. This rule applies only to cases which are neither very recent nor very protracted, *i. e.,* to the fully developed acute disease, since lymphocytosis is found in nontuberculous meningitis tending to recovery, in acute syphilitic meningitis, and in other chronic

*See Encephalitis Lethargica, p. 624.

brain affections, while polynucleosis is occasionally associated with lymphocytosis in chronic tuberculous meningitis.

Of interest *chemically* are the facts that in meningitis the proportion of chlorides in the cerebrospinal fluid is often reduced while that of albumin is increased. The albumin consists principally of serin, while normally it is mostly globulin. The fibrin is increased, while the reducing agent is often absent.

The *course of meningitis* varies greatly not only with the cause but with the clinical types of the affection and the severity of the epidemic as well. Some cases are mild and transient, "abortive"; others are extremely malignant, "fulminant," in nature, ending fatally within a day or two, or sooner. The mode of commencement offers no certain indica-
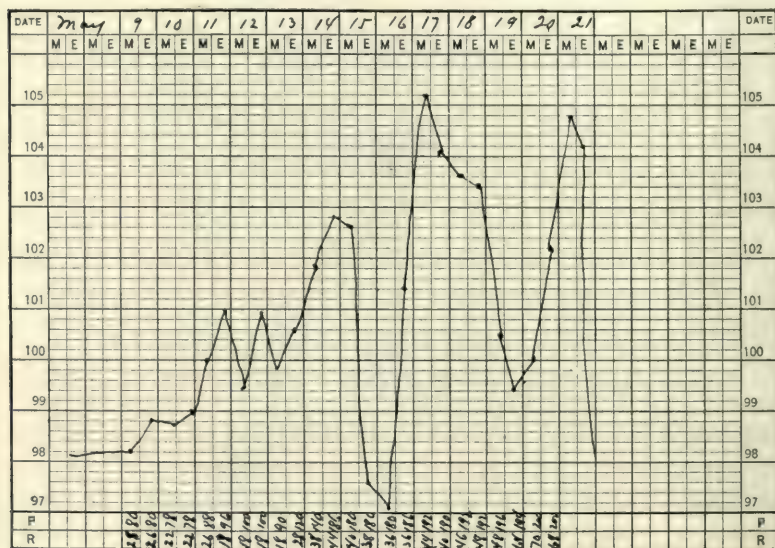
Fig. 176.—Fever curve of tuberculous meningitis in a child two years old.

tion as to the ultimate course. As previously mentioned, primary meningitis begins more suddenly and progresses more rapidly than the secondary variety. The great majority of cases are usually ushered in by profuse vomiting, rise of temperature, severe headache, pain in the back and limbs, sensitiveness of the vertebral column, rigidity and convulsions. The fontanelles in infants are distended, the bowels confined, the abdomen retracted (trough-shaped), and the urine scanty, often albuminous. During the early period symptoms of excitement of function prevail. The patient is delirious, shrieks (hydrocephalic cry), is very sensitive to noises and light, but very soon he passes into a state of

sopor which gradually increases in intensity. At a later period of the disease there is depression of function. The pulse and respiration which in the beginning are accelerated, later become irregular and slow, the somnolence deepens to coma, and various paralyses appear. The afore-mentioned eye symptoms are usually quite marked and involvement of the facial nerve pronounced. In disease of the base, all parts of the facial nerve may be involved; in that of the convexity, only the lower part may suffer. In hopeless cases deglutition also becomes affected; the coma increases, the patient can no longer be roused; the conjunc-tival reflex is abolished, the eyes are smeared with mucus or pus; the corneæ are hazy or ulcerated; the sphincters are paralyzed; and after lingering in this moribund state for another few days the patient is finally relieved of the agony by death. Milder, nontuberculous cases may gradually recover. In this event the disease is usually fol-lowed by very slow convalescence and frequently by deaf-mutism, aphasia, amaurosis, idiocy, etc. Meningitis sometimes runs a pro-tracted course, continuing for weeks with periods of marked im-provement, but finally ends fatally. These cases generally represent the chronic form of infantile meningitis, which is essentially a men-ingoencephalitis.

Differential Diagnosis

In the early stages meningitis may be confounded with typhoid fever, pneumonia, acute exanthematous diseases, uremia and eclampsia from other causes. In *typhoid fever* the vomiting is less persistent, diarrhea the rule, impairment of the sensorium less marked and more gradual in development, the spleen enlarged, the fever characteristic (step-curve), and the blood responding to Widal's reaction. *Apex pneumonia* partic-ularly may be mistaken for acute meningitis. In pneumonia the "cerebral" symptoms often clear up with the establishment of the signs of pulmonary consolidation or develop very late in the course of the dis-ease, the respiration ratio is increased and expiration is prolonged, and the temperature is evenly high. On the other hand, in meningitis, the nervous symptoms increase with time, respiration is irregular or ster-terous and inspiration prolonged and sighing, and the temperature va-riable. The differentiation between meningitis and a sudden attack of *uremia* is based principally upon the condition of the urine which should always be tested in case of doubt. The history also is very help-ful. *Eclampsia* caused by gastrointestinal intoxication, etc., or develop-ing during the onset of some febrile disease is apt to be mistaken for meningitis the first twenty-four hours only—until the alimentary canal

has been emptied, or the other causes of the eclampsia have become apparent.

Latent tuberculous meningitis may lead to many errors in the diagnosis. It may be confounded with severe remittent fever, encephalitis, syphilitic meningitis, and tumor of the brain. In *remittent fever* the plasmodium malariæ or pigment is readily found in the blood; *encephalitis* can be excluded by the absence of tubercle bacillus in the cerebrospinal fluid; in *syphilitic meningitis* there are other evidences of syphilis (choroiditis, rhagades, spirochete, etc.); in *tumor of the brain* the progress of the disease is slow, and there are permanent focal symptoms (localized paralyses, optic neuritis, etc.) to account for a local lesion. In doubtful cases lumbar puncture and the tuberculin reactions will materially aid in the diagnosis.

Bearing in mind the clinical signs and the findings in the cerebrospinal fluid there should be but little difficulty in differentiating the usual forms of meningitis with fair precision—at least so far as it pertains to the tuberculous or nontuberculous variety. Briefly stated the differential symptoms of the latter types are as follows:

DIFFERENTIAL DIAGNOSIS

TUBERCULOUS MENINGITIS	NONTUBERCULOUS MENINGITIS
History: Preceding indisposition	Apparent good health; infectious diseases or otitis
Temperature: Low in the beginning	High
MacEwen's sign: Pronounced	Slight
Cerebrospinal fluid: Clear; tubercle bacillus; lymphocytosis (mononuclear)	Cloudy or purulent; no tubercle bacilli; polynucleosis
The eyes: Optic neuritis; choroid tubercles	Absent
Skin eruptions: Indefinite	Frequently petechiæ
Paresis: Early and variable	Late
Von Pirquet's test: Positive	Negative
Complement-fixation reaction:* Usually positive	Negative

The prognosis at best is very grave. Tuberculous meningitis is invariably fatal. The mortality in nontuberculous meningitis ranges between 50 per cent and 75 per cent. Where operative procedures can be brought into use, *e. g.*, traumatic or otic meningitis with localized lesions, the outcome is more hopeful, provided no time is lost and the patient's general health is fair.

Treatment.—Aside from operative treatment wherever indicated, lumbar puncture for the relief of pressure symptoms, and meningococcic antitoxin, little need be expected from all other methods of

*See p. 84.

treatment in vogue. With the advance in our bacteriologic study of the cerebrospinal fluid and the possibility of early detection of the etiologic factor of the meningitis in question, there is reason to hope that the majority of cases of meningitis will be combated by a curative serum. Wonderful results are already on record from the early intra-spinal and intravenous use of antimeningococcic serum in meningitis due to the diplococcus intracellularis. (See p. 78.)

More recently some clinicians have claimed excellent results from the introduction of Flexner's serum directly into the lateral ventricle of the brain, after withdrawing the inflammatory exudate. This pro-cedure, of course, can only be employed in infants, where the ante-rior fontanelle is still open. (See p. 209.)

The symptomatic treatment consists of warm baths with or with-out mustard every three or four hours; ice bag to the head, bro-mides and stronger hypnotics to relieve excessive irritation; small doses of calomel and large doses of sodium iodide; careful nursing (feed-ing by mouth, gavage* or per rectum), and stimulation as necessity arises. Special attention should be paid to cleanliness of the mouth and nasopharynx, and avoidance of decubitus.

When an epidemic prevails, all such prophylactic measures should be instituted as are recommended for other contagious and infectious diseases, special care being taken to disinfect nasopharyngeal dis-charges. As a prophylactic urotropin also may be tried.

℞	Natrii Iodidi	℥ ss	2.00
	Natrii Bromidi	℥ i ss	6.00
	Aq. Menthæ Pip.	℥ iv	15.00
	Aq. Destil.	q. s. ad f ℥ ii	60.00
	M.		

S.—One teaspoonful every six hours, for a child three years old. (Routine treatment.)

℞ Hyoscin. Hydrobromatis   gr. $\frac{1}{500}$ to gr. $\frac{1}{300}$

S.—Hypodermically, for a child three to six years old. (To relieve excessive excitation.)

Diplegia Spastica Infantilis

(CONGENITAL RIGIDITY OF THE LIMBS, LITTLE'S DISEASE)

The nature of this form of infantile paralysis is still obscure. De-generative changes have frequently been found in the pyramidal tracts or their correlated structures of the encephalon. But whether they are the results of early antenatal arrested development (poren-cephalia), intrauterine brain disease, traumatism during labor (embol-

*By introducing through the nose a soft rubber catheter.

ism or hemorrhage) by instruments or dystocia; or prematurity, are questions awaiting a correct solution. Some cases are certainly acquired.

The symptomatology of this affection is sometimes manifested soon after birth and sometimes not until the child begins to walk. One of

Fig. 177.—Diplegia spastica infantilis in a baby eight months old who sustained cerebral injuries (with hemorrhages) during obstetric delivery. Note rigidity of neck and extremities (right arm is contracted and right leg pressing against left one): baby is unable to change its position without assistance. Note also convergent strabismus as a result of paralysis of the N. abducens.

Fig. 178.—Little's disease. "Scissors-gait" or cross-legged progression.

the earliest symptoms is rigidity of the limbs. The child usually lies motionless (does not kick) with the legs pressed against each other or one upon the other. He begins to walk late and with difficulty or may not walk at all. If he is able to walk, he takes short, rigid steps with the feet in tiptoe position (talipes equinus) and the knees pressed closely together, or crossing each other, sometimes half running so that at every step a fall seems imminent. The rigidity gradually grows worse, leads to fixed deformities and extends to the upper extremities and even the trunk. A Z-shaped deformity is often observed in the hand when the patient attempts to use it. Early in the disease

Fig. 179.—Diplegia spastica infantilis (Little's Disease). Note extreme spasticity of the muscles of the upper and lower extremities, and inability to stand erect without support.

the deformities disappear during sound sleep or deep anesthesia. The knee jerks are exaggerated, ankle clonus is generally present, atrophy is slight and develops late, and the sphincters are normal. The majority of cases present symptoms of defective psychical development (up to idiocy), stammering, nystagmus, strabismus, athetosis and epileptic convulsions. Where the latter symptoms prevail, the prognosis is very bad, otherwise it is not absolutely unfavorable.

Treatment.—Under suitable treatment the progress of the disease may be arrested and a partial cure effected. The treatment consists of stimulating baths, passive motion, educational exercises, massage and galvanization, and immobilization in the corrected position by suitable braces for a period of months. If this fails, we may resort to tenotomy, tenectomy, tendon transplantation, partial resection of the motor nerves (Stoffel's operation), and resection of the posterior nerve roots (Foerster's operation), followed by the aforementioned therapeutic measures. Antisyphilitic medication is sometimes beneficial. When the seat of the lesion in the brain is discovered *early,* an attempt may be made to ameliorate the otherwise hopeless conditions by trephining, and evacuation of blood clots,—decompression,—or removal of tumors, if there be any. Persistent and painstaking after-treatment is essential to success. (See p. 643.)

The differential diagnosis between this disease and polioencephalitis is based principally upon the absence in Little's disease of true paralysis and the presence of the characteristic, jerky, half-running, spastic scissors-gait.

Hemiplegia Spastica Infantilis

(Spastic Cerebral Paralysis, Polioencephalitis Strümpell)

The exact status of this diseased condition is still unsettled. Some authors look upon it as an irregular type of encephalitis (*q. v.*) or polioencephalitis (see "Poliomyelitis").

Anatomically, after abatement of the acute process (which consists of inflammation, hemorrhage, embolism and thrombosis in the gray motor cortical substance) it is, manifested by sclerosis, atrophy, fatty or cystic degeneration of certain portions of the brain—of several convolutions, an entire lobe, or of the large brain ganglia ("agenesis corticalis"). Not rarely the pyramidal tracts down to the medulla spinalis exhibit secondary descending degeneration.

It is a disease of early childhood, up to four years of age, and usually develops suddenly (very rarely insidiously), with fever, nausea, vomiting, headache and convulsions, or, less frequently in connection with other infectious diseases, such as exanthema, pneumonia, etc. After subsidence of the acute symptoms it is noticed that one-half of the body, or one arm or one leg is more or less paralyzed. The affected elbow hangs close to the body and the arm is bent to the ulnar side, while the fingers are flexed into the hollow of the hand; the foot is often distorted in an equinovarus position with the great toe overextended at right angles to the metatarsus. The patient walks practi-

cally on the toes of the paralyzed leg. As the disease progresses, the affected limbs become atrophied and contracted and the hand manifests a great tendency to athetotic and choreic movements. The tendon reflexes are exaggerated and there is more or less marked muscular rigidity. The muscles never exhibit reaction of degeneration. Sensation is unimpaired. The cranial nerves (facial and optic), as a rule, are involved, but not to a great extent. Sometimes there are also disturbances of speech (as a result of involvement of the hypoglossus), epilepsy, and mental impairment up to total idiocy. In the course of

Fig. 180.—Hemiplegia spastica infantilis, by some authors looked upon as a ''cerebral'' or ''encephalitic'' type of poliomyelitis with lesions chiefly in the motor area of the cerebral cortex. Note peculiar position of the right leg in the act of walking and characteristic ''athetotic'' hand.

time, especially under suitable treatment (which is practically the same as in anterior poliomyelitis) the paralysis, atrophy and contractures may somewhat improve and, in mild cases, disappear entirely; but on the whole the prognosis is bad. The patients are usually helpless in mind and body, are very prone to suffer from epilepsy and,

where the cerebral symptoms are pronounced, they rarely attain the age of twenty or thirty years.

As already suggested, this form of polioencephalitis may be mistaken for atypical encephalitis or anterior poliomyelitis. In both of these affections, especially in typical poliomyelitis, athetosis and spasticity of the extremities (both pathognomonic symptoms of spastic cerebral paralysis) are absent. Furthermore, in poliomyelitis there are reaction of degeneration and diminution or loss of tendon reflexes—the contrary being the case in the disease in question. This disease calls for further study for its clarification.

The treatment is essentially the same as in encephalitis, *q. v.*

Encephalitis

(Nonsuppurative, Hemorrhagic, Encephalitis)

Acute encephalitis is encountered principally in young children. It may be primary and occur either sporadically or in epidemic form, in the latter event often in connection with epidemic poliomyelitis.

Fig. 181.—Left hemiplegia following acute encephalitis. Note drooping of left shoulder and dragging of left leg in the act of walking.

Secondary encephalitis usually occurs in association with divers acute infectious diseases, such as influenza, diphtheria, scarlatina, pneumonia, pertussis, etc.

Any portion of the brain and medulla may be the seat of the inflammation, although there seems to be a predilection for the gray substance of the cortex. The changes in the brain consist principally of cellular infiltration of the vascular walls, perivascular cellular exudation, hemorrhage and thrombosis. The larger foci at first appear red and soft and later yellowish-white. After the process has run its course the affected part of the brain usually shows marked atrophy with cicatricial contraction.

The clinical course varies with the seat and extent of the brain lesion. As a rule it begins suddenly with nausea, headache, vomiting, high fever and often convulsions. This is followed by stupor, slow pulse, Cheyne-Stokes' breathing, constant deviation of the eyes to one side; and if the medulla is involved, also by implication of some cranial nerves, e. g., facial and hypoglossal. In infants the fontanelle is usually not bulging. As a rule the tendency of acute encephalitis is towards recovery, except for remaining mono- or hemiplegia, and often mental impairment (see Hemiplegia Spastica Infantilis, p. 618).

Treatment.—The treatment is symptomatic. Ice bag to the head, warm baths, bromides and liquid nourishment. Lumbar puncture is of but little therapeutic value.

Acute encephalitis may be mistaken for acute cerebrospinal or tuberculous meningitis. The differential diagnosis must be based principally upon the cytologic findings in the cerebrospinal fluid (see p. 611). The absence of bulging of the fontanelles in infants points strongly against meningitis, more especially of the tuberculous variety. For its differentiation from polioencephalitis see pp. 620, 639.

Brain Abscess

(Encephalitis Purulenta)

Suppurative encephalitis most frequently develops in connection with inflammatory or suppurative processes in adjacent structures, e. g., the eyes (panophthalmitis), the nose (caries of the cribriform bone), and especially the ears (mastoid disease). It also occurs as a result of traumatism, foreign bodies in the brain, pyemia, pulmonary abscess or gangrene, ulcerative endocarditis and embolism.

The encephalitis may be diffuse or circumscribed, run an acute or chronic course. The classical brain abscess is the *chronic* variety. Pathologically, this term should be limited to circumscribed collec-

tions of pus in the brain surrounded by a yellowish-white, rather dense, newly formed membrane, possessing all the characteristics of a pyogenic membrane. It is not to be confused with an *acute* brain abscess in which definite lines of demarcation from the healthy tissue are absent. Congruent with circumscribed abscesses of other portions of the body, the inner layer of the membrane lining the pus cavity is formed of soft granulation tissue, while the contiguous structures are edematous, reddened and highly vascular. Brain abscesses may be single or multiple, and if multiple and of long duration may become confluent and attain considerable size. The pus in acute abscess is reddish or yellowish in color, while in chronic abscess the pus has a greenish-yellow color and a consistence similar to synovial fluid. It is acid in reaction. Unless contaminated by necrosis of the bone or foreign bodies the pus is usually odorless. An encapsulated abscess after remaining stationary for a considerable time shows a tendency to extend, not gradually, but in steps. According to Bergmann, each step represents a new inflammation, and at autopsy one finds the traces of a recent softening adjacent to some portion of an older abscess cavity. Large portions of the brain may thus be destroyed, and if the gray matter is preserved, an abscess may extend over the whole lobe or even throughout an entire hemisphere without producing definite symptoms indicating the trouble. The meninges rarely escape involvement.

The clinical picture of purulent encephalitis is very misleading and varies greatly with the seat and extent of the lesion and the stage of the disease. It is less confusing in cases of cranial traumatism, but, even in as severe an injury as fracture of the skull, the cerebral symptoms may be so vague as for days to escape notice. The onset is usually sudden with nausea, vomiting, fever, stupor, and convulsions. Older children complain of dizziness and headache. This condition may last one or two days or as many weeks. Then either the coma increases and is followed by death, or the symptoms abate, and the patient is apparently on the road to recovery, except that in the majority of instances monoplegia, or hemiplegia with or without involvement of some cranial nerves is left behind. The subsequent course of the disease depends upon the nature of the brain lesion.

Suppurative encephalitis of very limited extent, with its cause removed, may clear up without appreciable after effects. On the other hand, where an encapsulated abscess has formed, the violent symptoms may abate and the acute pass into a chronic stage. This state reached, the encephalitis is apt to run a very protracted course; with recurrent violent exacerbations and deceptive remissions; on the one hand, giv-

ing rise to symptoms of acute meningitis; on the other, especially if the abscess is large and pressing upon the motor areas and cranial nerves, to those of tumor of the brain. In either case the diagnosis is often extremely difficult. Ordinarily *meningitis* differs from abscess in that it pursues a more acute course, and the brain symptoms are indicative of a more diffuse lesion. The diagnosis between *brain tumor* and abscess is much more difficult. In abscess there is usually an irregular temperature with rigors, motor aphasia and paraphasia, while in tumor fever is rare and there is a greater tendency toward disturbances in the area of distribution of the cranial nerves at the base of the brain, and toward choked disc. (See "Brain Tumor", p. 645.) A history of ear disease or direct violence points strongly toward abscess. Slowly developing focal brain symptoms are characteristic of brain tumor. These differential points, however, at best, are not very reliable.

As previously mentioned, the remissions occurring during the course of chronic brain abscess are very deceptive. In the first place, the "latent period" is rarely entirely free from signs of ill health. As a rule, the patient suffers from occasional headache, vomiting, rise of temperature, mild paresis, etc. Secondly, there is no way of telling when in the midst of apparent good health the abscess may suddenly rupture in the brain ventricles or meninges and rapidly end fatally.

Treatment.—The prognosis of brain abscess, therefore, is always very grave, unless surgical interference is resorted to early. The operative results are especially favorable in abscesses due to otitis or trauma—provided they can be localized.

Purulent encephalitis before operation, should be treated by perfect rest, ice bags to the head, lumbar puncture, etc.—the same as acute meningitis.

Early prophylactic measures, especially energetic treatment of ear trouble, scrupulous attention to suppurative conditions of the eyes, nose and throat are all powerful in the prevention of the dreadful complication and sequelæ.

To emphasize the difficulty encountered in diagnosing chronic brain abscess even under the most modern methods of observation, I may relate the following interesting case:

H. D., aged four years, 27 lbs. in weight. Family history good. Parents living and well, have four additional healthy children. Child was normal at birth, breast fed, and free from any serious illness until two years of age, when he had an attack of measles, apparently mild in character. One year later he began to complain of headache and occasional vomiting. This condition continued for several months, notwithstanding careful care and treatment, and a stay at Mt. Sinai Hospital for

ten days. He came under our observation at the end of November. We made a tentative diagnosis of tuberculous meningitis. Admitted to the Postgraduate Hospital (H. D. Chapin's service) December 1st. Temperature for following week ranged between normal and half a degree above, pulse between 70 and 92, and respiration between 24 and 28 per minute. Child moaned and complained of headache. Persistent projectile vomiting, especially after breakfast. Staggering gait, ataxia of right arm. Choked disc in both eyes, more marked in left. Tuberculin and Wassermann reactions negative. The same result of examination of cerebrospinal fluid and urine. *Blood* shows 90 per cent hemoglobin, 5,392,000 erythrocytes, 24,000 leucocytes, 36 per cent lymphocytes and 64 per cent neutrophiles. *Roentgen-ray* examination discloses the following picture: Pituitary fossa enlarged and base eroded; glenoid processes atrophied; also atrophy of anterior portion of vault—all pointing to tumor of pituitary gland. *Operation,* January 5. Escape of large quantity of cloudy fluid, free from tubercle bacilli. No growth about cerebellum and nothing found after incision in right lobe. An ulcerated spot about ½ inch in diameter is visible on superior surface of vermis. Puncture of this area fails to bring pus. The boy promptly recovered from the effects of the operation and improved for about ten days. Gradually grew worse thereafter; developed a higher temperature, from 101° to 105° F. and died January 27. *Autopsy:* Large ulcerated area, size of silver dollar, in summit of vermis, purulent collection under the membrane about pons and crura. Incision of cerebellum reveals an abscess cavity size of thumb and almost 1½ inches in length, occupying the right lobe and extending slightly to the left lobe. Pus shows no bacteria in pure culture.

Whether the abscess was the direct result of the measles or the consecutive latent otitis could not be determined.

Lethargic or Epidemic Encephalitis

(Meningo-, Encephalo-, Myeloneuritis)

Though supposedly of very recent origin, this affection has undoubtedly occurred on previous occasions, and either passed unnoticed or was diagnosed as nonsuppurative encephalitis or the cerebral type of poliomyelitis, with both of which diseases it has several symptoms in common. There are two definite records which substantiate this view. An epidemic of "sleeping sickness" occurred in 1712 in Tübingen (Germany) and its vicinity. The other record refers to an epidemic of encephalitis which prevailed in 1890 during and after the influenza epidemic in Austria-Hungary, Italy and Switzerland, and was then spoken of as "Nona." The most recent epidemic of lethargic encephalitis dates back to 1916, and was first described by von Economo of Vienna. Since then numerous cases have appeared in divers parts of Europe and America, following the trail blazed by the destructive epidemics of influenza, justifying the assumption either that this affection acts as a predisposing cause of encephalitis, or that the same infectious agent forms the etiologic factor in both affections. Von Wiesner,[1]

[1]Wien. klin. Wchnschr., p. 933, 1917.

J. A. Wilson,[2] and Strauss and Loewe[3] present evidence to the
contrary, yet, until further preponderating corroboration has been
adduced, the question of the exact identity of the causal factor is best
left in abeyance.

Pathology.—Whatever its identity, recent observations have shed
considerable light on the mode of activity of the infectious agent. It
has been shown to attack the central and peripheral nervous system
and its coverings in a very widespread manner. The structures par-
ticularly involved are those about the third ventricle, the acqueduct
of Sylvius, the lateral ventricles and optic thalamus, and the pons and
medulla. Occasionally lesions are found also in the cortex and in the
cerebellum. The spinal cord also is subject to attack. The lesions are
of an inflammatory, sometimes hemorrhagic, character, and occur in
nodular and diffuse forms.

Microscopically we find thickening of the leptomeninges with exu-
dation or vascular congestion. The gray matter is the site of peri-
vascular cellular infiltration. There is ample evidence of toxic de-
generation of the nerve cells and neuronophagy.

Symptomatology.—The symptoms correspond, of course, with the
functions of the cerebrospinal system affected. Thus, if the lesion is
localized in the globus pallidus, tremor and rigidity result; if in the
thalamus: choreiform athetotic movements; if in the meninges: rigidity;
if in the spinal cord: neuritic pain; if in the cranial nerve nuclei: facial
paralysis, ophthalmoplegia, etc.; and finally if the cerebellum is attacked,
ataxia supervenes. In our opinion it is erroneous to speak of special
types of the disease, as the symptomatology may at any moment
undergo considerable modification with extension or retrogression of
the inflammatory process.

In children the onset of the affection is rather sudden, with rise of
temperature, vomiting and more rarely, convulsions. Sometimes the
attack is preceded by sore throat, lassitude, and headache. The fever
usually remains moderate during the entire course of the disease, only
exceptionally reaching 104° or 105° F. The pulse is generally rapid,
and, as will be mentioned later, may become very irregular. About
twenty-four hours after the onset it is noticed that the patient is
losing interest in his surroundings, becomes drowsy and apathetic,
and, as time goes on, the lethargy becomes so deep and continuous as
almost to resemble a state of coma. Yet with some effort the patient
may be sufficiently aroused to respond to questions and to partake
of nourishment. During the profound lethargy the child's face as-

[2]Quart. Jouf. Med., Oxford, p. 88, 1918.
[3]Jour. A. M. A., p. 1373, 1920.

sumes a mask-like appearance (Parkinsonian); it is pasty, waxy and motionless. Some children are disturbed in their slumber by sharp pain in the face, arms and legs, and when aroused present marked choreic or athetotic movements of the head and arms. In the great majority of cases there is more or less pronounced involvement of the cranial nerves. Most common by far is oculomotor and abducens paralysis, with ptosis, diplopia and ophthalmoplegia externa and interna. Next in frequency is unilateral or bilateral facial paralysis. More rarely, the glossopharyngeal and vagus are affected, as may readily be determined by the impairment in speech and difficult deglutition, respiratory and cardiac arrhythmia, hiccough, etc. In a number of cases, and usually late in the course of the disease, rigidity of the neck, Kernig's and Brudzinski's signs are present, and where the motor areas are involved, monoplegia, hemiplegia and diplegia supervene. Occasionally, also, anesthesias and paresthesias and spontaneous muscle spasms are encountered.

Diagnosis.—In view of the multiplicity of the symptomatology the diagnosis, in the absence of an epidemic, presents considerable difficulty. Lethargic encephalitis may be mistaken for nonsuppurative encephalitis (see p. 620), complicating or following divers acute infectious diseases; cerebrospinal meningitis, tuberculous meningitis and polioencephalitis. Lethargic encephalitis is characterized by progressive stupor, early involvement of the cranial nerves, especially the oculomotor (ptosis, etc.); paralysis of the extremities, late if at all; mask-like face; neuritic pain and choreiform movements or tremors. In *simple encephalitis* the cranial nerves are affected late, whereas mono- or hemiplegia appears early; the Parkinsonian expression of the face, the choreic movements and pain are usually absent. In *cerebrospinal meningitis* opisthotonos, Kernig's and Brudzinski's signs appear early and are very marked, and there are several other symptoms in meningitis which are absent in lethargic encephalitis. In *tuberculous meningitis*, the onset is usually slow, paralysis and optic neuritis occur early; Babinski's reflex and McEwen's sign are marked, tubercle bacilli are present in the cerebrospinal fluid and there are in addition other symptoms of acute meningitis. In *polioencephalitis* paralysis of the extremities appears early, while the mask-like face and profound stupor are absent. According to Barker, Cross and Irwin,[*] a cell count in the cerebrospinal fluid of from 10 to 100 small mononuclears along with a positive globulin reaction, a negative Wassermann and absence of tubercle bacilli or meningococci,

[*]Am. Jour. Med. Sc., March, 1920.

at a time of an epidemic of encephalitis, point strongly to the existence of this disease.

Prognosis.—As a rule, the course of the disease is protracted, extending over many weeks, although occasionally mild as well as severe cases are encoutered which are on the road to recovery in a few days. On the other hand, fulminant cases of epidemic encephalitis are met with which may end fatally in but a few hours. The death rate is lower in children than in adults, and ranges between 10 to 20 per cent. Involvement of the sphincters, hyperpyrexia and progressive stupor are of grave import!

Convalescence is usually slow and occasionally interrupted by slight relapses. The possibility of sequelæ in the form of mental deterioration and epilepsy, should not be lost sight of.

Treatment.—Absolute rest to body and mind is essential during the entire course of the disease. Liquid diet; where deglutition is difficult milk and broths may be given by gavage, with a catheter introduced through the nose. This may be repeated twice or thrice daily. In the early stages I believe to have obtained great benefit from wet cups applied to the nape of the neck. From 4 to 6 ounces of blood is withdrawn once or twice. The temperature and pain are best relieved by warm baths, with or without mustard. In symptoms of brain pressure, especially where several cranial nerves are involved, lumbar puncture is quite useful. Netter* recommends the induction of a fixation-abscess by a subcutaneous injection of 1 c.c. of turpentine. Hexamethylenamine is worth trying, especially in the early stages of the disease.

During convalescence prolonged rest and quiet preferably in the country. Nutritious diet. Massage and hydrotherapy. Hematinic tonics.

Poliomyelitis Anterior

(Polioencephalitis, Poliomyeloencephalitis, Infantile Paralysis)
(Heine-Medin Disease)

Our knowledge of poliomyelitis has been slow and gradual in its evolution notwithstanding the fact that two score or more epidemics† of the disease have offered unusual facilities for its careful study.

---

*Bull. de l' Académie de Méd., Vol. 83, No. 13, 1920.

†In modern times the following great epidemics of poliomyelitis have been recorded. In 1905, in Norway and Sweden, together 2000 cases. In 1907 the first great epidemic occurred in America, 2500 cases being reported in and around New York. In 1909 there were outbreaks in various parts of the United States and Cuba with a total of 2,343 cases. In 1910 an epidemic of infantile paralysis spread almost throughout the entire country, about 500 cases occurring in the District of Columbia, Iowa, Massachusetts, Minnesota, Indiana, and Pennsylvania, and about 400 cases in Maryland, New Hampshire, New York, Rhode Island,

The first scientific essay on the subject was written by J. Heine in 1840. Herein he attributes the affection to a lesion in the spinal cord. In 1851 Rilliez and Barthez contested this view and designated the disease as "Essential Paralysis of Children." In another contribution on the subject, in 1860, Heine reasserted his opinion, but failed to meet with authoritative support, until, in 1870, Joffroy and Charcot announced that they found distinct changes in the spinal cord consisting of "primary involvement of the ganglion cells leading to atrophy." Thereupon "Essential Paralysis" was replaced by "Spinal Paralysis in Children," or, in short "Infantile Paralysis." In 1872 Duchenne called attention to the loss of reaction in the paralyzed muscles to the faradic current, and four years later Erb demonstrated absence of reaction also to the galvanic current. Our knowledge was further advanced by Seeligmüller by furnishing an instructive contribution to the study of the pathogenesis of the contractures and deformities following poliomyelitis. All the while every trifling ailment and mishap were blamed for the origin of the disease in question; and although in 1884 Strümpell suggested that an infectious agent must play an active rôle in the causation of the affection, we still note that as late as the year 1893 no less an authority than Gowers relates several cases of poliomyelitis which he thought were due to catching cold from sitting on wet grass. Medin is deserving the credit for having systematized the symptomatology of infantile paralysis—in 1890—and we are indebted to Wickman for developing —in 1907—the epidemiology of the disease and for classifying it into several distinct types. Our knowledge of the etiology of poliomyelitis was greatly enhanced—in 1909—by Landsteiner, Popper, Flexner and Lewis, who demonstrated experimentally that monkeys are

Virginia, Washington and Wisconsin. The epidemic of 1916 exceeded all previous epidemics in severity as well as in the number of cases, in New York State alone over 13,000 cases having been reported. The total must assuredly have been much larger, since a great many mild and so-called abortive cases must inevitably have escaped attention. A large number of cases have recently reappeared in Boston and vicinity and about 100 cases in New York.

POLIOMYELITIS IN BOSTON

Week Ending	Total Cases	Total Deaths	Non-resident Cases	Non-resident Deaths	Resident Cases	Resident Deaths	Massachusetts Cases
July 24, 1920	1	1	0	1	1	0	4
July 31, 1920	8	0	2	0	6	0	10
August 7, 1920	5	2	0	0	5	2	5
August 14, 1920	15	4	2	1	13	3	16
August 21, 1920	13	3	6	1	7	2	25
August 28, 1920	14	3	3	0	11	3	26
September 4, 1920	22	9	7	3	15	6	52
September 11, 1920	26	3	13	2	13	1	53
September 18, 1920	29	4	9	2	20	2	66
September 25, 1920	30	4	10	3	20	1	68
October 2, 1920	27	3	12	2	15	1	72
October 9, 1920	11	1	1	0	10	1	53
October 16, 1920	11	3	3	1	8	2	46
Totals	212	40	68	16	144	24	496

susceptible to this affection, and, furthermore, that in these animals one attack of paralysis prevents a second successful inoculation; in other words, it produces an immunity against the disease. Further studies, moreover, established the fact that in human beings also one attack immunizes against another one, and that the serum of recovered monkeys as well as men contains a specific substance which is capable of neutralizing the virus *in vitro*. This neutralizing agent was shown to exist also in the blood of a large number of so-called abortive cases.

Etiology.—With these facts in view an entirely new light was thrown upon the mode of dissemination of the disease, since it became immediately obvious that poliomyelitis, like so many other communicable affections, is transmitted by an infective agent that follows the lines of human contact and travel, and is carried not only by the victims of the disease, but by virus-carriers as well. Experimental and clinical evidence is gradually accumulating which tends to show that the virus of poliomyelitis enters the human body most frequently, even if not exclusively, through the upper respiratory tract and is carried to the cerebrospinal system by means of the lymphatics.

Owing to the not infrequent occurrence of paralysis among lower animals, *e. g.*, chickens and dogs ("distemper"), some authors thought it plausible to fasten the source of infection to this agency, but careful investigations undertaken during the 1916 epidemic by the Federal and States Boards of Health, with the assistance of expert veterinarians, utterly failed to substantiate that assumption. Moreover, it was conclusively shown that in fowl, for example, the paralysis was the result of peripheral rather than central nerve lesions. There is much more scientific basis for the supposition that the disease may be conveyed by flies, since it has been repeatedly demonstrated by Flexner and Clark among others that the common house fly can carry the virus of poliomyelitis in a living and actively infectious state for forty-eight hours or longer, and abounds during the period of greatest prevalence of the disease, *i. e.*, the hot summer months. Now, if we accept the hypothesis of transmission of poliomyelitis by insects, more especially flies, then the probability of conveyance of the disease to the human body by means of food contaminated by house flies and the like holds true with equal force. Be it remembered, the virus of poliomyelitis withstands both low degrees of cold as well as ordinary degrees of heat for long periods of time, and when enclosed in albuminous matter it resists drying for several weeks. In view of the aforesaid and the fact that the greatest number of victims of the affection are met in children under three years

of age* whose diet consists principally of milk, this article of food must naturally come under the suspicion of being the purveyor of the infectious agent of poliomyelitis. Yet, after a very thorough investigation of the subject in question, the Committee of the Department of Health of the City of New York has arrived at the conclusion that food, and milk in particular, plays no part in the transmission of the disease. We must add, however, that this exhaustive investigation notwithstanding, we would err greatly in ignoring the aforementioned hypothesis so far as prophylaxis is concerned, at least until such time as the identity of the infectious agent is definitely established. Unfortunately thus far all bacteriologic researches have failed to demonstrate the etiologic factor of poliomyelitis microscopically. It is therefore generally assumed that it is not bacterial in character, but belongs to the group of the so-called ultramicroscopic filtrable viruses. Experimentally it has been shown to be highly resistant to diverse destructive measures. It withstands glycerination for long periods of time and is not affected by 0.5 per cent of carbolic acid; it is but slightly influenced by freezing at 2 to $-4°$ C. for forty days; the virus is less resistant to high degrees of heat—it can be destroyed by a temperature of from $45°$ to $50°$ C., if exposed for half an hour. It can be destroyed also by a 2 per cent solution of peroxide of hydrogen, by methol and by corrosive sublimate.

Pathology.—During the last two decades, particularly, great advances have been made in the study of the morbid anatomy of poliomyelitis. Whereas originally the opinion generally prevailed that the lesions of this affection were essentially limited to the anterior horns of the spinal cord, it is now definitely settled that no portion of the cerebrospinal system may escape involvement, and, moreover, as is the case of other grave communicable diseases, the lesions are frequently disseminated throughout various other structures and organs of the body. Since the upper nasal cavities are in direct communication with the meninges by means of the lymphatics which pass outward with the filaments of the olfactory nerve, and since the earliest changes are noticeable in the perivascular lymph spaces of the blood vessels of the leptomeninges, it seems reasonable to conclude that the virus enters the human body through the upper respiratory tract. Microscopically the meninges are usually found injected and edema-

*Of 5,346 cases of poliomyelitis tabulated by the N. Y. City Board of Health during the 1916 epidemic, the age incidence was as follows:

6 months or younger	192 cases	6 years	245 cases
1 year	793	7 years	160
2 years	1,398	8 years	127
3 years	1,998	9 years	78
4 years	693	10 years	56
5 years	412	10 to 15 years	94

tous, and the brain and cord moist, translucent and edematous. The gray matter of the cord is also swollen and projects above the level of the white matter. Minute hemorrhages are often distinguishable in both the gray and white matter, the former often assuming a grayish-pink hue. The cerebrospinal fluid is but little increased. Microscopically the pathologic process is found to consist chiefly of a cellular exudation, hemorrhages and edema. The lesions are most pronounced where there is an abundance of blood vessels, hence in the cervical and lumbar enlargements, more particularly in the anterior horns of the cord and in the medulla. ''The cellular exudate forms a sheath apparently completely surrounding the vessels for long stretches and in places the cells are so numerous as to form thick collars which seem to press on the lumen and thus exert a mechanical effect in obstructing the circulation'' (Peabody, Draper and Dochez). A similar mechanical as well as toxic action is progressing in the intimal lining of the blood vessels, the conjoint pressure soon leading either to hemorrhagic softening or anemia-pressure-necrosis of the infiltrated structures and gradual replacement of the ganglion cells by cicatricial tissue. Of course, this terminal pathologic stage is usually not reached where the pressure is early relieved by absorption of the hemorrhage and cellular exudate; hence, the large number of mild and so-called abortive cases, and the tendency towards spontaneous recovery. In recording his observations on human and experimental poliomyelitis Howe distinguishes three pathologic types of the disease: (1) Cases in which the lesions are limited to infiltration of the pia and blood vessels: the mesodermic tissue type; (2) cases in which the main feature is degeneration of the motor cells in the anterior horn, accompanied by the proliferation of neuroglia; the ectodermic tissue type; and (3) the mixed type. The first group represents the general reaction of the organism to the infection, manifested by changes in the central nervous system and the lymph tissues of the body. In the second group the changes in the central nervous system of man are polymorphous. The reaction in the ganglion cells and nuclei allows the recognition of no less than eight different forms in the degenerative process consequent to the poliomyelitis infection. The mixed type is usually encountered in human poliomyelitis. As already stated, the virus of poliomyelitis is productive also of extensive pathologic changes in the lymphoid tissues and parenchymatous organs. Peyer's patches and some of the mesenteric glands show lesions resembling those observed in typhoid fever. The superficial glands of the body, the tonsils, the thymus gland, the liver and occasionally the spleen are considerably enlarged. The affected muscles show definite signs

of degeneration. Some of their fibers disappear entirely and others
are shrunken, the whole limb being atrophied as a result thereof.
Often the bones participate in this pathologic process.

Symptomatology and Course.—An affection based upon so vast and
varied morbid anatomy must obviously manifest itself by an equally
as complex a symptomatology, ranging between that of simple, local
and often transient paralysis, and general, frequently fatal, toxemia.
No wonder that prior to our full understanding of its pathology al-
most every type of the affection was described as a separate clinical
entity, a disease *sui generis*. For that matter even the present tend-
ency to classify poliomyelitis into several distinct types is hardly
justifiable from a pathologic point of view; and having had the op-
portunity to observe a great many cases during the last two epidemics
and at other times, the author cannot help but feel that no one classi-
fication will cover all cases clinically. Hence our reason for not attempt-
ing to present one.

Initial Stage.—After an incubation period lasting from three to
twelve days, and towards the end indicated by indefinite symptoms
of ill health, such as slight fatigue, irritability and anorexia, the tem-
perature all at once rises, up to 104° F., the child complains of ir-
regular, muscular pain, headache and sore throat or other symptoms
of old fashioned grip or is seized with an attack of indigestion, with
diarrhea and sometimes vomiting, in young children not rarely ac-
companied by convulsions. Physical examination reveals diffuse
congestion of the throat, with or without a slight grayish deposit upon
the tonsils, slight rigidity of the neck, especially on bending the head
towards the sternum, marked paresthesia, muscular jerking or tre-
mors, distinct drowsiness, and irritability when disturbed. The mind
is usually clear even in grave cases. The heart's action is generally
exaggerated, even when the fever is low. These symptoms may remain
stationary for from twenty-four to seventy-two hours and then show
a tendency towards spontaneous abatement (*abortive type*) or get
rapidly worse—herald the advent of paralysis.

Paralytic Stage.—The paralysis usually sets in insidiously, is often
preceded by progressive muscular weakness and either remains
localized or swiftly spreads to other parts of the body, the degree of
severity and extent of the paralysis depending, of course, upon the
gravity and seat of the lesion. In the majority of cases, especially
during mild epidemics, the pathologic process is limited chiefly to the
spinal cord (*spinal type*). In this event the paralysis usually involves
the extremities alone, or, less frequently, the neck, abdomen, spine or
chest as well. The paralysis may be partial or total. The extremities

are usually affected in the following order of frequency: one leg, both legs, one arm, both arms, one leg and one arm on opposite sides or more rarely on the same side, both legs and one arm, both legs and both arms, and both arms and one leg. Occasionally the paralysis remains limited to a group of muscles or even to a single muscle, *e. g.*, the tibialis anticus, gastrocnemius, or deltoid, and is not rarely overlooked until atrophy has set in. When the muscles of the neck are implicated, the child is unable to hold the head erect; the latter drops (neck drop) either for-

Fig. 182.—Poliomyelitis "spinal type;" lesion in lumbar enlargement; atrophy and right "drop-foot."

Fig. 183.—Poliomyelitis "spinal" type; lesion in cervical enlargement; paralysis of upper arm as well as right serratus magnus, "angel wing" deformity of right scapula, marked muscular atrophy.

ward or backward, or sways from side to side. In paralysis of the abdominal muscles, owing to active intraabdominal pressure by gases, there is "ballooning" of the affected muscles which contrasts strongly with the flatness of the intact muscles. With the spinal muscles affected the patient shows a peculiar clumsiness in turning around or from side

to side while lying flat on his back, and is unable to assume a sitting posture without assistance. This paralysis is ordinarily overlooked until frank scoliosis has made its appearance. Sometimes the paralysis manifests itself in stages, at intervals of several hours, so much so, that occasionally the muscles implicated first may already be on the mend while a new group of muscles may just about be attacked. Where the lesions are limited to the lower neuron the paralysis is flaccid in character, the tendon reflexes greatly diminished or lost, the reaction to the

Fig. 184.—Poliomyelitis "spinal type;" lesion in cervical and dorsal regions; partial paralysis of the muscles of the neck, abdomen, and right thigh (atrophy).

Fig. 185.—Poliomyelitis "spinal type;" lesion in cervical enlargement; "neck drop."

faradic current lost, while that to the galvanic current may persist for some time. Sensation is but slightly impaired. There is no tendency to acute decubitus.

In a small percentage of cases the paralysis, beginning with the lower extremities, gradually spreads upward (*progressive* or *ascending type*),

resembling Landry's paralysis), involves the upper extremities, the external muscles of respiration, and the diaphragm, if the lesion reaches the upper part of the cervical cord. In this event exitus may take place after from two to four days as a result of respiratory failure. On the other hand, the paralysis may start in the arms and from here spread downwards (*descending type,* resembling transverse myelitis) to the

Fig. 186.—Poliomyelitis affecting the abdominal muscles giving rise to "ballooning" of the abdomen.

Fig. 187.—Poliomyelitis "bulbospinal type;" lesion in medulla; paralysis of left facial nerve, left forearm and left leg.

lower extremities. In these cases we usually find paralysis of the vesical and anal sphincters, giving rise to urinary retention or dribbling and obstinate constipation or incontinence of feces, respectively.

In another group of cases the inflammatory process extends to the medulla (*bulbospinal type*). The lesion is generally unilateral, excep-

tionally bilateral, and clinically characterized by partial or total paralysis of some of the cranial nerves, in addition to the manifestations observed in the purely spinal variety of poliomyelitis. As a rule, the facial and abducens are affected, less frequently the glossopharyngeal and vagus, and occasionally also the hypoglossal nerve, in which event the patient presents not only facial paralysis, inward strabismus, and more or less marked respiratory difficulties (Cheyne-Stokes' breathing, cyanosis

Fig. 188.—Poliomyelitis "pontine" or "cerebral" type; lesions in pons, medulla, and spinal cord; paralysis of right facial nerve, left forearm and hand, external respiratory and abdominal muscles and right leg.

and cardiac arrhythmia), but also disturbance of phonation and deglutition. These cases are usually very grave, nay, often fatal within a few days. In the absence of concomitant paralysis of the extremities one is apt to diagnose laryngeal diphtheria. Indeed, on several occasions the author was invited to intubate these cases. Where the cord remains intact and the lesion localized in the medulla alone, the tendon reactions are usually exaggerated, the limbs more or less rigid, and there is a

distinct tendency towards ataxia (*ataxic type*).* The aforementioned symptoms are much more pronounced where the pathologic process invades also the pons (*pontine type*), and the condition is further aggravated by the usual concurrence of oculomotor paralysis which may lead to complete ophthalmoplegia, and cross paralysis or hemiplegia alternans.

During the recent epidemics ample evidence was brought forth to

Fig. 189.—Same case as Fig. 188 showing also high degree of scoliosis.

prove that the so-called primary polioencephalitis (Strümpell, see Hemiplegia Spastica Infantilis), instead of being a distinct clinical entity, is probably a cerebral or encephalitic type of poliomyelitis. As is well known, this type of the disease is manifested by the predominance of meningeal symptoms, such as recurrent explosive vomiting, convulsions, rigidity of the neck up to opisthotonos, and marked stupor. Kernig's

*Some authors attribute the ataxia to a lesion in the cerebellum; the postmortem findings, however, do not substantiate this claim.

and Brudzinski's signs are usually inconstant and appear late, and seem to be due rather to the resistance on the part of the child to the painful flexion of the spine. After a day or two partial or complete spastic paralysis of one or several extremities supervenes, not rarely accompanied by involvement of the facial nerve. In some cases there is also marked incoordination of the extremities. The tendon reactions are usually greatly exaggerated.

In the majority of cases pain, either spontaneous or on passive motion, forms a conspicuous symptom of acute poliomyelitis. As the pain often follows the course of the nerves, as in neuritis, these cases are sometimes grouped in a separate class—the *polyneuritic type*. According to Lovett, the pain and tenderness are sometimes marked enough to cause the paralysis to be entirely overlooked, and a diagnosis of rheumatism or scurvy to be made. In 2 cases under our observation during the last epidemic hip-joint disease was diagnosticated.

Prognosis.—As already stated a great many children fail to survive the acute phase of the affection. The mortality seems to vary with the virulence of the epidemic. Thus, whereas in the Massachusetts epidemic (1907-10) of 1,599 cases only 125 died, the epidemic of 1916 destroyed 3,310 young lives in New York State out of a total of 13,177 victims of poliomyetitis.* The highest death rate, about 63 per cent, occurred among the cases in which the lesions extended to the medulla and pons, most frequently either as a result of respiratory failure in consequence of paralysis of the respiratory muscles, or secondarily to complicating bronchopneumonia. Most of them, about 80 per cent, succumbed during the first week of the onset of the disease, only 11 per cent in the second week, 3 to 4 per cent in the third week and about five per cent some time later, as a result of exhaustion and complications. The highest mortality was noted in children under five or over fifteen years of age, higher among males than females.

Convalescent Stage.—This stage starts with the subsidence of the acute symptoms, such as pain and fever, and with the permanent arrest

*MOVEMENT OF CASES, DEATHS AND FATALITY RATES FROM POLIOMYELITIS DURING THE EPIDEMIC OF 1916 IN NEW YORK STATE, BY MONTHS†

MONTH	State of New York			New York City			Rest of State		
	Cases	Deaths	Fatality Rate per 100 Cases	Cases	Deaths	Fatality Rate per 100 Cases	Cases	Deaths	Fatality Rate per 100 Cases
June..............	367	64	17.4	313	53	20.1	54	1	....
July..............	4,011	895	22.3	3,443	779	22.6	568	116	20.4
August............	5,987	1,466	24.5	3,927	1,080	27.5	2,060	368	17.9
September.........	1,992	628	31.5	985	364	37.0	1,007	264	26.2
October...........	645	215	33.3	258	122	47.3	387	93	24.0
November.........	135	40	29.6	47	25	53.2	88	15	17.0
December.........	40	20	50.0	18	11	61.1	22	9	40.9
Total...........	13,177	3,310	25.1	8,991	2,444	27.2	4,186	866	21.1

†M. Nicoll, Jr. (New York State Med. Jour., Vol. xvii, No. 6).

of the paralysis. It corresponds with the stage when the excessive exudate in the brain and cord is getting absorbed, the pressure upon the vital structures is being spontaneously relieved to a greater or less degree, and consequently some of the paralyzed nerves or muscles begin to functionate. The degree and extent of the initial paralysis is no criterion as to the final outcome of the disease as a whole. The author has watched many children, seemingly in a hopeless condition, to recover almost completely, and *vice versa*, some apparently mild localized paralyses to persist for life, notwithstanding most scrupulous and scientific treatment. The muscles that fail to recover within about ten days after the acute attack promptly begin to show signs of atrophy (the limb is flabby cold and cyanotic). Associated with the atrophy is reaction of degeneration. The response of nerve and muscle to the faradic current is usually lost, while the galvanic irritability persists, sometimes for a year or two after the onset of the affection. Owing to the laxity of the muscles and their inability to hold the articular ends of the bones in apposition, the joints soon become the seat of subluxations. As the paralysis continues, the trophic changes become more and more marked—the limbs lose their shape, often look like mere skin and bone, and the growth of the bones becomes retarded. Moreover, owing to the activity of the intact, antagonistic muscles, sooner or later divers deformities make their appearance. In cases where all the muscles of an extremity are uniformly involved, the limb remains free from deformity, but is limp and lifeless and hangs attached to the trunk like an artificial limb.

Permanent Stage.—The paralysis may be looked upon as permanent, if the case fails to improve after two years' careful treatment. Reaction of degeneration of the nerves and muscles is usually complete, and the deformities (talipes, scoliosis, etc.) are fully established. The deformities are generally less pronounced in the so-called cerebral type of poliomyelitis.

Diagnosis.—Typical, spinal, poliomyelitis (*i. e.*, sudden, more or less complete, flaccid paralysis of one extremity or several of them, or of a group of muscles of the trunk, preceded by moderate fever and other symptoms of an ordinary cold or indigestion) usually presents no diagnostic difficulties, whether or not it is met with during the prevalence of an epidemic. If pain forms a conspicuous symptom, poliomyelitis may in the initial stage be mistaken for scurvy, rheumatic fever, or polyneuritis. Now, in scurvy we generally find a history of a slow onset; tumefactions along the long bones, ribs and the bones of the head; sponginess and bluish, hemorrhagic discoloration of the gums, and the immobility of the extremities is due to fear of pain and tenderness but not

to actual paralysis. This latter symptom is characteristic also of rheumatism. Besides, in this affection the pain is more acute and localized and usually associated with some swelling, especially about the joints. Furthermore, rheumatic fever is not rarely complicated by chorea and endo- or pericarditis. Polyneuritis is very uncommon in young children; as a rule, it follows metallic poisoning or serious infectious diseases, is most apt to begin with the extensor muscles of the hands and feet, and the symmetrical paralysis does not recede as early as the paralysis of poliomyelitis. During an epidemic of infantile paralysis diverse tuberculous and traumatic affections of the bones and joints frequently lead to diagnostic errors; however, in doubtful cases a Roentgen-ray examination and tuberculin test will readily clear up the diagnosis. Much more difficulty is encountered in interpreting correctly the other types of poliomyelitis, more especially in the absence of an epidemic. Thus, the pontine and cerebral types have several symptoms in common with acute meningitis and secondary encephalitis. But on closer observation it will usually be noted that stupor, Kernig's and Brudzinski's signs appear earlier than in poliomyelitis and are also more marked and more constant. On the other hand, the paralysis appears earlier and is more extensive, as a rule, in poliomyelitis. Furthermore, secondary encephalitis follows or complicates some infectious disease, e. g., influenza, pneumonia or scarlatina. As errors in the diagnosis may prove instrumental in spreading the affection to all others coming in contact with the patient, it is wise, where there is the least doubt, to proceed with a careful examination of the cerebrospinal fluid. According to Peabody, Draper and Dochez, who have made an exhaustive study of poliomyelitis, the cerebrospinal fluid taken during the early days of the disease, and especially before the onset of the paralysis, as a rule, shows an increased cell count with a low or normal globulin content. At this early stage the polymorphonuclears may amount to 90 per cent of the total cells. Later, however, most fluids show almost exclusively lymphocytes and large mononuclear cells. After the first two weeks the cell count usually drops to normal, or nearly normal, and there is frequently an increase in the globulin content. Analogous changes may be found in the spinal fluid of abortive cases. All fluids examined by those authors reduced Fehling's solution. As the cerebrospinal fluid of poliomyelitis greatly resembles that of tuberculous meningitis, it is advisable to exclude the presence of tubercle bacilli in the former. Where further confirmation of the diagnosis becomes necessary, we may resort also to the colloidal gold reaction of the cerebrospinal fluid, which according to Felton and Maxcy is constant and positive in the acute stage of poliomyelitis.

While the blood picture of patients suffering from poliomyelitis is not as specific as the spinal fluid, it is nevertheless of some diagnostic value if taken in connection with other available evidence. There is usually a leucocytosis of from 15,000 to 30,000, and the polymorphonuclear cells are increased at the expense of the lymphocytes.

For the differential diagnosis between polioencephalitis and lethargic encephalitis see p. 625.

Treatment.—Prophylaxis.—With the earliest detection of suspicious signs of acute poliomyelitis, the patient should be promptly isolated, and handled in the same manner as other communicable diseases (see p. 68). During an epidemic, vomiting, fever, headache, diarrhea, congestion of the throat, rigidity of the neck and drowsiness, should be looked upon as suspicious of poliomyelitis. When the diagnosis has been confirmed the attendant should be quarantined together with the patient for about three weeks. If for financial reasons this proves impracticable, it is advisable to remove the patient to a suitable hospital. All discharges from the mouth, nose and throat should be received on cloths or toilet paper and immediately burned. The feces and urine should be disinfected prior to their disposal. The room of the patient must be screened to keep out flies, mosquitoes and other insects. Before lifting the quarantine, the clothing, bedding, utensils, etc., of the patient should be disinfected, and the sick-room and its contents thoroughly cleaned and aired. All those known to have come in contact with the patient should be carefully watched—for about twelve days—for the aforementioned suspicious signs of poliomyelitis, and if need be, promptly isolated. During the period of observation children should not be permitted to attend school for about two weeks. Cleansing of the nose and throat twice daily with antiseptic solutions, *e. g.,* dioxide of hydrogen 2 per cent, is worth trying, although it has recently been shown that antiseptics may irritate the nasal mucous membrane and render it more susceptible to bacterial invasion. We may try also the internal administration of hexamethylenamine, as a preventive of poliomyelitis, since it has been proved to find its way in the cerebrospinal fluid and to exert a germicidal effect. From 10 to 15 grains daily, in divided doses, will usually suffice. Whenever possible, individuals should occupy beds singly.

Active Treatment.—1. Acute Phase.—Absolute rest and quiet to body and mind is essential during the acute course of the disease. The patient should be kept in bed, in recumbent posture, for about ten days, and the affected limbs immobilized, even after apparent recession of the paralysis, to prevent early muscular contractures and deformities. This is easily accomplished by the application of light

splints, well padded with wadding, to the paralyzed limbs. The feet should be supported at right angles to the legs, and in cases where the spinal muscles are involved, it is best to put the patient in a Bradford frame. As in all febrile affections the diet should be nutritious and easily digestible, and should consist of broths, boiled milk, fruit juices, and well-cooked cereals. Where deglutition is difficult, cautious feeding by stomach tube may have to be resorted to.

No specific has thus far been discovered to combat poliomyelitis in any of its forms or stages. Immune serum, supposedly efficient in preventing or arresting the progress of poliomyelitis in monkeys, has as yet failed to show any appreciable benefits in human beings. Nevertheless, for want of more effective therapeutic measures, its use should be encouraged, especially in grave cases. If utilized, we must be sure that the donor is free from syphilis. The serum is administered in the same manner as antimeningitis serum, by lumbar puncture and intravenously. It should be injected on three successive days in doses of from 15 to 20 c.c. The serum is valuless after the acute stage. In rare cases intraspinal injection of serum is followed by a reaction meningitis. As in other acute cerebrospinal affections, lumbar puncture is a sovereign remedy also in poliomyelitis, where symptoms of brain pressure manifest themselves. It may be employed once or twice daily, according to indications. Of medicinal agents, urotropin, sodium salicylate and sodium bromide, of each from 3 to 5 grains every four hours will generally be found useful. Respiratory and heart failure should be treated with oxygen inhalations, and camphor and strychnine or caffeine hypodermically. The author believes that beneficial results are obtained from the administration of potassium iodide, in from 2 to 5 grain doses every four hours; he assumes that the iodides aid in the absorption of the cellular exudation and thus relieve intraspinal pressure. Severe headache may be mitigated by an ice bag to the head. High fever may be reduced by warm baths, which are also indicated in excessive cerebral irritation. Subdural injections of suprarenal solutions have thus far proved of no material benefit, and the same is true of intravenous injections of salvarsan.

2. Convalescent Stage.—After subsidence of the acute symptoms and complete cessation of the pain and tenderness, an inventory, as it were, should be made of the stationary damage to the nerves and muscles inflicted by the highly destructive virus. As a rule paralysis in some form is left behind. Where the paralysis is partial or limited to single muscles, the ''spring balance muscle test'' may have to be resorted to, to determine with any degree of exactitude, how much power there is still left in the affected muscles. This test, by the way,

is also of great value to register in pounds, at certain intervals, the gain or loss in muscular strength after a certain method of treatment. The concensus of opinion of the profession is at present in favor of getting the patient in a sitting and, if possible, in an upright position as soon as possible, provided the paralysis is not very extensive. Of course, this should be done only with the aid of suitable braces, to prevent deformities. Where the spinal or abdominal muscles are implicated, support should be furnished by means of an accurately fitting light corset, and in cases where the lower extremities are effected, the so-called caliper splint should be applied. Where the glutei are also involved, we have to resort to a walking frame and light crutches. In paralysis of the deltoid the arm should be supported in a sling, and to prevent permanent deformities of the forearm, the latter is put in a well-padded wire splint. The less burdensome the splints, etc., the better. Furthermore, it is very important not to fatigue the patient, whatever method of treatment is adopted.

To prevent early atrophy and to improve the impoverished circulation of the structures involved, massage, including vibration, heat, electricity and muscle training, including bath exercises, are of undoubted therapeutic value. The treatment should begin after the pain and tenderness, spontaneous as well as on passive motion, have completely ceased. The massage should be gentle, local as well as general, and should be applied once or twice daily for about twenty minutes at a time. Later, the massage may be supplemented by light vibratory muscular stimulation. The patient should be very warmly dressed, and the affected limb should in addition be exposed daily, for ten minutes at a time, to dry heat obtained either from a large electric bulb or the numerous baking apparatus on the market. The benefits derived from the use of electricity have been grossly exaggerated; yet a mild faradic and galvanic current, applied for from five to ten minutes at a time, every other day, may hasten recovery by inducing mild muscular contractions, by improving nutrition and promoting conduction of nerve impulses. Muscle training or passive and active motion corresponding to the normal muscular action, is the *sine qua non* in the restoration of the muscular functions, but it requires a very thorough familiarity with the exact powers of each muscle or group of muscles. Otherwise by exercising the muscles in the wrong direction considerable harm will be done. Bath exercises also are very beneficial. It will sometimes be noted that where patients show no muscular power in an extremity, when put into the bath they are able to demonstrate some power in those muscles,—the buoyancy of the water apparently overcoming the gravity of the limb. As the entire cooperation and con-

centration of attention of the patient is indispensable to its successful performance, muscle training is only applicable in children over five years of age. Furthermore, this mode of treatment is best entrusted to an expert in this line of work.

A number of clinicians claim to have obtained excellent results from the injection of strychnine in the paralyzed muscles. This treatment was originally recommended by Charcot. He administered, once daily, 1/40 to 1/50 grain. As strychnine in small doses is a useful general tonic, it can do no harm and possibly may do some good. It may advantageously be combined with the glycerophosphate of iron. General supportive treatment, ample, nutritious food and fresh outdoor air are excellent adjuvants in the reestablishment of the dormant bodily functions.

3. Permanent Stage.—If after giving the aforementioned methods of treatment faithful trial without any appreciable benefit to the patient, but on the contrary the paralysis persists and the deformities become fixed, there is nothing else left but to attempt to correct the deformities by operative procedures. The profession is not agreed on the time when an operation becomes indispensable. Some surgeons advise waiting two years, others twice as long or even longer. Hence it is best to leave the decision of this important question to the good judgment of the individual surgeon. As to the choice of the particular operations, R. W. Lovett offers the following suggestions:

Talipes Equinus.—Stretching, tenotomy of the tendo Achillis, if the anterior muscles have fair power. Transplantation of the extensor of the great toe or other extensors into the tarsal bones, anterior silk ligaments with or without tenotomy, tenodesis, arthrodesis.

Talipes Calcaneus.—Astragalectomy, tenodesis, arthrodesis.

Talipes Varus.—Transplantation of the anterior tibial, when that is active, to the outer third of the foot. Silk ligament from the fibula to the cuboid; astragalectomy, tenodesis, arthrodesis.

Talipes Valgus.—Transplantation of one of the peroneals to the inner side of the foot, silk ligaments from the tibia to the inner side of the tarsus; astragalectomy, tenodesis, arthrodesis.

Flexed Knee.—Stretching or open division of the hamstrings.

Hyperextended Knee.—In cases where the quadriceps is paralyzed and the hamstrings and the gastrocnemius are good, transplantation of one or two hamstrings into the tubercle of the tibia.

Knock-knee.—Supracondyloid osteotomy (Soutter's operation).

Flexed Hip.—Fasciotomy, if severe.

Dislocated Hip.—Arthrodesis.

Shoulder.—Dropping of the arm away from the glenoid cavity, arthrodesis of the joint, silk ligaments.

In cases of deltoid paralysis with the pectoralis major active, the origin of the latter may be transplanted into the spine of the scapula.

The operations on the forearm, elbow and wrist vary greatly in individual cases. Arthrodesis of the elbow is useful, but the operation is not applicable at the wrist on account of the nature of the joint.

Scoliosis.—Treated in the same manner as scoliosis due to other causes than poliomyelitis.

It is essential to the success of these operations to select a surgeon who is thoroughly familiar with this work. But even in the best hands, the results are not invariably good. This is especially true of cases which have been greatly neglected or treated by the numerous quacks who thrive upon the ignorance of the unfortunate people.

Tumors of the Brain

Of the total number of cases of brain tumors on record about one-half occurred in children. It is more common in boys than in girls. The usual seat is in the cerebellum and the basal ganglia. Brain tu-

Fig. 190.—Secondary passive hydrocephalus in tumor of the brain. (O. Vierodt, Pfaundler and Schlossmann.)

bercle is especially common, and relatively frequent also are divers forms of sarcoma (gliosarcoma). These are often metastatic. Hidden as intracranial neoplasms are from sight and touch, their nature must necessarily be a matter of conjecture only, except, perhaps, in cases of bony growths, which may be diagnosed by means of the Roentgen-ray, and tubercle and syphilis which may be surmised by the presence of other tuberculous or syphilitic lesions in other parts of the body or detected by the tuberculin, complement fixation, or Wassermann tests.

The diagnosis of brain tumor is based upon the general and local nerve disturbances they produce. As a rule, the general symptoms precede the local, and consist of headache, vomiting, vertigo, optic neuritis, and convulsions.

The headache is usually persistent, but may also be periodical, suggesting a malarial origin.. The headache may be frontal, vertical or occipital, or equally distributed over all parts of the cranium. The locality of the pain occasionally bears a direct relation to the seat of the tumor, thus, when the growth is in the white substance the pain is usually frontal; when beneath the tentorium, occipital, etc. The same rule often applies to the pain elicited on tapping the skull over the seat of the disease. Intense headache in infants is indicated by rolling of the head from side to side, by throwing up the hands to the head, contraction of the eyebrows, and intolerance to light. The headache is frequently followed but may also be preceded by vomiting.

The vomiting is projectile in character, and comes on suddenly. It differs from gastric vomiting by the absence of other signs of stomach trouble, and from vomiting accompanying migraine by that the headache does not always terminate with it. Vomiting is especially characteristic of tumor in the medulla oblongata or the middle lobe of the cerebellum, but it may occur in tumors affecting any part of the brain.

The vertigo may be constant or paroxysmal and is most marked in affections of the pons or cerebellum. Vertigo in infants frequently escapes notice. It is manifested by sudden drooping of the head, pallor of the face and occasionally also vomiting.

Optic neuritis sometimes forms one of the earliest symptoms of brain tumors. It does not always correspond to the size of the tumor. The choked disc is usually bilateral. It may develop slowly or rapidly, and in either case the optic neuritis proceeds to complete optic atrophy.

The child's nervous system being highly susceptible to irritation, increased intracranial pressure is quite early productive of convulsions of varying severity. The convulsions may be general or local. General convulsions with loss of consciousness may occur in tumors of any part of the brain, but are more common in tumors of the posterior fossa than in those of the anterior or middle fossa. Local convulsive seizures are met with chiefly when the neoplasm occupies certain situations. For example, convulsions beginning in the foot, as a rule, are indicative of the lesion being in the upper region of the motor area; those of the arm, the middle region; and those of the

face, the lower region. It should be remembered, however, that the effects of a tumor may extend far beyond its actual site, and, furthermore, as the case proceeds, convulsions, which from the outset have been local, may become general. The convulsive attacks may recur frequently and last from several seconds to as many hours. The convulsions are not rarely followed by paresis or paralysis of the affected limbs. At first the muscular weakness may be transient, but as the disease advances it becomes permanent.

The focal symptoms of brain tumors are also manifested by uni- or bilateral hemiplegia, monoplegia, affections of speech, and paralysis of cranial nerves. (See "Brain Localization" p. 602.) The local symptoms pointing to the seat of the tumor attain their greatest precision when the swelling—be it a new growth or an inflammatory mass—is seated in the motor area of the cortex. They do not always correspond, however, to the size of the tumor. Furthermore, as the brain usually accommodates itself gradually to the increasing pressure and functional interference produced by the new growths, the appearance of the focal symptoms is frequently delayed until a very late stage of the disease. Once established, local symptoms are of great help in arriving at a correct diagnosis, except, perhaps in cases where the tumor is multiple and distributed through various parts of the brain. (See "Tuberculosis of the Brain," p. 452.)

Diagnosis.—With the determination of the seat of the tumor, the diagnosis is greatly facilitated but rarely entirely settled. Brain tumors have several symptoms in common with tuberculous and syphilitic meningitis, brain abscess, epilepsy and hysteria. The differentiation between tuberculous and syphilitic tumors, and chronic tuberculous and syphilitic meningitis is extremely difficult and often impossible especially when the tumors are multiple. In *tubercle* and *gumma* the symptoms are more gradual in development, the optic atrophy more pronounced, and the focal symptoms more marked and localized, while the course of tuberculous or syphilitic meningitis is much more rapid and, besides, there are several other symptoms pathognomonic of meningitis. In doubtful cases some valuable information may be obtained from the tuberculin and Wassermann tests and from a careful examination of the cerebrospinal fluid. In *acute brain abscess* optic atrophy is absent, there is usually a history of acute infection, ear disease, or trauma, and the symptoms of purulent encephalitis, such as chills, fever, stupor, etc. In the absence of this history there is practically no way of distinguishing *latent chronic abscess* from tumor, as has already been emphasized on a previous occasion (p. 623).

Jacksonian epilepsy may resemble brain tumor in its early stage, but as the disease advances the diagnosis can readily be cleared up by the absence of optic neuritis and other focal symptoms. There are cases on record of *hysterical* hemiplegia with convulsions and contractures which were mistaken for brain tumor. Careful investigation, however, will usually reveal the absence of optic neuritis, and the fact that in hysteria the symptoms are inconstant and multifarious, rather sudden in development, and rarely progressive in character.

The nature of the tumor can sometimes be established by its seat. Thus, if the tumor is located in the cerebellum or pons, it is probably tubercle or glioma; if in the cortex, it is apt to be syphilitic. Cysticerci are most commonly met with in the meninges or cortex. Abscesses are usually situated in the cerebral or cerebellar "hemisphere," and but rarely in the central ganglia, the pons, medulla, or the middle lobe of the cerebellum. Too much reliance, however, cannot be placed upon these observations.

Treatment.—In view of the possibility of the tumor being syphilitic, it is always advisable to put the patient on an active antisyphilitic course of treatment (the iodides and mercury). In syphilitic disease prompt treatment will soon be followed by amelioration of the symptoms, and, if faithfully persisted in, often by a cure. This therapeutic measure is occasionally attended by favorable results also in growths other than syphilitic, and should, therefore, be resorted to as a routine procedure in all obscure brain lesions. Antisyphilitic treatment proving negative, and tonics, in the form of fresh air, generous diet, cod liver oil, iron and the hypophosphites, failing to benefit the patient,— tonic treatment may do well in tubercle, and if employed early may in exceptional cases arrest its growth,—the question of surgical interference should be taken under advisement. An operation is indicated where the tumor is single, and situated superficially in a part of the brain (motor area of the cortex) which can be reached and from which the tumor can be removed without immediate danger to life. Under favorable conditions, an operation should be performed early, before the general health has greatly suffered and permanent injury has resulted to organs and limbs from persistent brain pressure. Recently successful attempts have been made to remove growths from deeply seated structures; the results as to life and good health, however, are still too few and too far between to warrant precipitate action.

In hopeless cases morphine and its derivatives will help to relieve the agony. (For "Tumors of the Pituitary Gland," see p. 570.)

Epilepsia

(EPILEPSY; FITS)

Epilepsy is an obscure affection of the brain, in typical form characterized by attacks of loss of consciousness, local or general convulsions, and a great tendency toward psychic disturbances. The situation and exact nature of the brain lesion is still undetermined, but, judging from the pathologic alterations (atrophy, hypertrophy, abscess formation, sclerosis, porencephalia, retention of subcortical cells, changes in the blood, etc.) so frequently found postmortem, there is reason to believe that there is no one pathologic entity responsible for the morbid condition.

The causes of epilepsy are many and divers. Congenital defects of the brain or skull; traumatism to the brain or skull (during birth or after); infectious diseases affecting the brain directly or indirectly; toxemias of all kinds, including grave gastrointestinal intoxication; repeated attacks of convulsions from reflex causes; neoplasms, including syphilitic and tuberculous; sudden psychic disturbances, such as sudden shock, etc., among many other as yet obscure causes, all contribute their share toward development of epilepsy at some period of life. An hereditary disposition is traceable in a certain number of cases, and children of syphilitic, alcoholic, and neurotic parents are more prone to contract the affection than those free from such encumbrances.

No age is exempt from the disease, but it is most apt to develop in children of from two to fifteen years old.

The exact time of the beginning of the disease cannot always be traced, since the symptoms may be so mild as to escape observation. The child may for a few moments ''hang its head,'' turn pale, and the paroxysm would be over with—hardly any reason to suspect epilepsy. The little attack may not recur for weeks or months, so that the last one is long forgotten when the next one sets in. It is only after the attacks grow longer in duration, stronger, more frequent, are preceded by an aura and possibly followed by involuntary urination and defecation, and profound sleep, that the nature of the dreadful condition is fully realized.

Genuine epilepsy varies greatly in severity not only in different individuals but also at different times. In addition to the rudimentary forms later to be described, the paroxysms are generally classified into severe (*grand mal*), mild (*petit mal*), and cortical or *Jacksonian*. The attacks are frequently preceded by a warning (*aura*) of motor, sensory or vasomotor character. There may be slight twitchings

of the limbs, eyes, or head, slight general tremor, a vague sensation in the stomach, a feeling of numbness or pricking in the extremities, hearing of noises, seeing of colors or sparks, smelling of peculiar odors, irritability, hallucinations, etc.

In **grand mal** immediately following the aura, and also without it, the patient, who may appear to be in good health, suddenly cries out, loses consciousness and falls, and becomes fixed in a tonic spasm, with face and limbs contorted and breathing suspended. His face is pale or cyanotic; his eyes are widely open (pupils usually dilated) and staring or rolled upward or sideward. The teeth are pressed firmly together, with the tongue often impacted between them. In a moment the fixed spasm gives way to clonic convulsions. The face, body and extremities twitch violently, and the head beats strongly backward. During this stage the face is congested and often bathed in perspiration. Foam frequently fills the mouth, and may be mixed with blood from the severely bitten tongue. As the contractions cease, the child sinks down exhausted, limp and lifeless—except for deep sighing respiration—into a state of profound sleep (postepileptic coma) of variable duration. With return of consciousness he has no knowledge of what occurred. The duration of the paroxysms varies between one and five minutes. It may occur once or several times a day, a week or month, or may not return for several months and even years. A certain periodicity, however, is demonstrable in a great many cases. The attacks may also occur at night, during sound sleep.

Petit mal is usually manifested by sudden loss of consciousness of very short duration. The patient may turn pale, stare vacantly, twitch a little, drop what he is holding, and then recover himself. Often in the midst of play the child suddenly stands fixed, "as if bewitched," with staring, absent-minded facial expression; a few moments later he resumes his play as though nothing had happened, or sinks down feebly or runs toward some object or person to support himself. The transition (sometimes after years) of petit mal into grand mal is not rare, and should always be remembered in fixing the duration of epilepsy.

In another group of cases the convulsions begin in one particular muscle or group of muscles, and rapidly spread to other parts of the body. Loss of consciousness may be absent or occur after the convulsions have become general. It is often followed by localized paresis. This *cortical* or *Jacksonian* form of epilepsy is based upon a definite local lesion in the cortex; it is acquired, whereas general convulsions may be both of prenatal or postnatal origin.

Epilepsy is not always represented by so typical a clinical picture. Rudimentary forms are encountered, which may tax the skill of even the best observer in reaching a correct conclusion.

In children as in adults instead of typical or atypical attacks of morbid physical phenomena, momentary states of mental disturbances may occur which may vary from simple confusion up to acute mania.

These fits occasionally alternate with convulsive seizures. Less common than in adults are the so-called postepileptic, frequently rather proepileptic, psychical aberrations which are manifested by unconscious, automatic, more or less violent actions, lasting minutes, hours or days. Inexplicable disappearance of children from home is not rarely an epileptic manifestation.

Epilepsia nutans ("Salaamkrampf") is manifested by sudden lightning-like spasmodic forward movements* (between 20 and 100) of the upper part of the body—a sort of reverential bow—and is associated with partial or complete loss of consciousness.

Epilepsia procursiva is characterized by a sudden forced start of running, of variable duration, which may cease abruptly or end in an attack of convulsions. Consciousness is partially lost during this seizure.

Epilepsy sooner or later leads to permanent mental impairment. In the earlier stages this may consist only of weakness of memory, silliness, alteration in the behavior (the child may be cranky, quarrelsome, destrucive, etc.), but as the disease becomes chronic the patient's mental dulness increases and may reach a state of total idiocy (see p. 751). Furthermore, with the growing mental hebetude there is also a corresponding development of coarse features with a downcast, dazed, and stolid facial expression—physical peculiarities which to the keen observer often betray some hidden central lesion. This observation often serves well in the differential diagnosis between epilepsy and reflex and hysteroid convulsive paroxysms. (See Spasmophilia and Hysteria.)

Treatment.—The termination of epilepsy is subject to great variations. With the recent gradual improvement in the methods of diagnosis and treatment, complete recovery from genuine epilepsy is far from being exceptional. This refers particularly to cases due to reflex causes (defective vision, adenoids, worms, phimosis, etc.), when detected early and remedied. To a great extent this is true also of cases resulting from traumatism or benign neoplasms, which are nowadays operated upon more or less successfully. The surgical results are especially gratifying in the Jacksonian form of epilepsy. Operative interfer-

---

*Similar forward movements are frequently observed in divers forms of idiocy.

ence, however, should always be preceded by an antisyphilitic course of treatment, which not rarely acts admirably. Some cases of epilepsy after resisting all sorts of "cures" for a number of years get well as unexpectedly as they got sick. Others again persist for life, do what you may. This is the so-called idiopathic epilepsy, for which from time immemorial the whole pharmacopeia, witchcraft, mental healing, Christian or unchristian Science, etc., have been used in vain. What can be accomplished, however, in such cases is the lessening of the severity and frequency of the attacks. All sources of irritation, however trifling, should be removed. The patient should be placed on a light, salt-free diet (milk, bread, cereals, vegetables, custards; eggs, fish; occasionally well-boiled meat; plenty of fruit and water) under the best possible hygienic conditions, and in the most congenial and restful surroundings. Residence in the country, with plenty of outdoor air, moderate exercise and hydrotherapy are ideal adjuvants.

If preceded by an aura sometimes in advance of the fit, the latter may occasionally be aborted by inhalation of amyl nitrite.

Immediate attention should be paid also to the convulsive fit, not alone to prevent a fatal issue from cerebral hemorrhage, or possibly from apnea, but principally to avoid grave bodily injury which the patient is apt to sustain during a severe fit. When the attacks are of frequent occurrence the child should not be left alone, especially in a room with an open fire, or in the vicinity of ponds, rivers, railroad tracks, etc., lest he be suffocated, fall out of bed, set himself on fire, drown, etc. A handkerchief or cork should be placed between the child's molar teeth to prevent biting of the tongue. A severe convulsive seizure may be aborted or modified by a few whiffs of chloroform, or amyl nitrite.

Of all remedies thus far recommended the bromides are the only ones which have proved of actual benefit in all forms of epilepsy. We should begin with moderate doses that will control the paroxysms. The bromides may advantageously be combined with small doses of Fowler's solution of arsenic. The treatment should be continued, with brief intermissions, to avoid bromism, for years—long after cessation of the attacks.

℞ Natrii Bromidi		
Ammonii Bromidi	āā ℥ ii	8.00
Strontii Bromidi	℥ i	4.00
Liquor Potassii Arsenitis	℥ ss	2.00
Mist. Rhei et Sodæ	℥ ss	15.00
Syr. Aurantii	q. s. ad f ℥ iii	90.00
M.		

S.—One teaspoonful in water every six hours, and later only twice a day, for a child six years old.

In severe fits we may add small doses of codeine.

When the bromides are not well tolerated by the stomach they may temporarily be administered per rectum. Postepileptic outbreaks frequently yield to early administration of hypnotics, especially chloral. Of late considerable success has been claimed from the administration of luminal (phenylethylbarbituric acid) in 1 gr. to 2 gr. doses, once or twice a day. It is supposed to be a non-habit-forming hypnotic and free from other deleterious effects. It is prescribed instead of or alternating with the bromides.

Migraine, Hemicrania

(Sick Headache)

There is reason to believe that the seat of the irritation upon which depend the pain and other manifestations of hemicrania lies in the brain (in the cortex or deeper cerebral structures). Cerebral hyperemia or anemia seems to be the immediate cause of an attack. The remote causes are very numerous. Gastrointestinal autointoxication seems to play a prominent rôle, and eyestrain, nasopharyngeal abnormalities, dental caries, helminthiasis, infectious diseases, and general debility are often found to act as predisposing causes. The disease prevails chiefly among nervous children over eight years of age, in girls more frequently than in boys. To some extent it seems to be hereditary.

Similar to epilepsy, migraine shows a distinct periodicity and is frequently preceded by premonitory signs, consisting of depression, irritability, visual disturbance, tremor, nausea and vomiting. The child complains of violent headache, usually along half of the head (hemicrania) or occiput. The pain is increased by jars, light, and noises, may last several minutes, hours, or days, and frequently terminates with an attack of vomiting followed by sound sleep, from which the patient awakes very much refreshed and apparently perfectly well. A prolonged attack is not rarely accompanied by psychic disturbance and even slight convulsions, in which event it may resemble organic brain disease, e. g., tuberculosis of the brain. The paroxysms may return after weeks, days, or months, at all events the disease runs a very chronic course, especially if no energetic efforts are made to determine the underlying cause and to remove it.

Treatment.—Where the cause cannot be detected or removed, a great deal of benefit is usually derived from improvement of the general health, especially attention to existing anemia, constipation, etc., and regulation of diet. Dilute hydrochloric acid (5 drops, well di-

luted, after each meal) often acts very beneficially. Sojourn in the country.

During an attack the patient should be kept quiet in bed, in a dark, well-ventilated room. Local moist heat, and caffeine and quinine (in cerebral anemia), and phenacetin and ergot with sodium bromide (in cerebral hyperemia) are of service to relieve the intense pain.

℞	Natrii Bromidi	℥ i	4.00
	Antipyrinæ		
	Caffeinæ Natrii Benzoatis	āā ℥ ss	2.00
	Syr. Aurantii q. s. ad f ℥ ii		60.00
	M.		

S.—One teaspoonful every six hours, for a child six years old.

Pavor Nocturnus

(NIGHT TERRORS)

Night terrors are observed chiefly in nervous children of from three to eight years old. Probably frightened by a horrible dream (seeing ferocious animals, etc.), the child suddenly awakes, jumps up, sits up in bed or jumps out, looks around staringly and anxiously, cries or screams for help, or utters incoherent words. After a few minutes he recognizes those about him, quiets down and falls asleep. The attack may recur once or more times a night or at longer intervals, and ultimately disappears (sometimes not until puberty) without serious consequences. In rare instances pavor nocturnus forms a precursor of epilepsy. I am inclined to think that the immediate cause of the attack is a cerebral hyperemia.

As a rule, the attack is aggravated by overloading of the stomach before retiring, faulty feeding, hearing of fearful stories, or seeing exciting shows, the presence of intestinal worms, adenoids and hypertrophied tonsils, and other local disturbances, and usually ceases upon removal of the aforementioned causes. The patient should sleep in an airy, slightly illuminated room, on a hard mattress, lightly covered and free from tightly fitting night clothes. The general health should be improved by outdoor air, cod liver oil, and other tonics. A moderate dose of sodium bromide at bedtime is useful to check frequently recurring attacks.

Syringomyelia*

Cavities in the cord may occur primarily as a congenital arrest of development or secondarily as a result of a gliomatous process in

---

*For "Congenital Malformations of the Spinal Cord," see p. 197.

the gray (cervical enlargement) and white matter. In pronounced noncongenital cases it is manifested by gradual loss of power in the upper limbs, trophic disturbances in the skin, subcutaneous tissue, and bones (glossy skin, ulceration and necrosis of the phalanges). disturbance of sensibility (partial or complete loss of pain- and temperature-sense while the muscular and tactile senses are preserved). Later, signs of muscular atrophy—beginning with a small muscle of the hand and gradually extending up to the shoulder—and paralysis, first of the upper then of the lower extremities, set in. The course of the disease is slow and occasionally interrupted by stationary periods.

Spinal Hemorrhage

(Apoplexia Spinalis, Hematomyelia)

The hemorrhage may be outside the dura, in the membranes, or in the substance of the cord. It is usually of traumatic origin—instrumental delivery, a fall or blow, or severe convulsions. The history of the case, therefore, is valuable in the diagnosis. Slight hemorrhage may give rise to no definite symptoms. The diagnosis of severe hemorrhage is based upon the sudden appearance of intense pain in the back, rigidity of the spine, sometimes convulsions, blood in the cerebrospinal fluid, and, if the pressure upon the cord is marked, paralytic symptoms. (See "Myelitis," p. 656.) The latter are especially pronounced in hemorrhage into the gray substance of the cord. Where the hemorrhage is moderate and the patient survives the immediate attack, the tendency of the affection is toward recovery. This may be enhanced by absolute rest on the face or side in a somewhat prone position. Local abstraction of blood; ice to the seat of injury. Later attention to the palsy. Cases due to fractures and direct violence must be treated surgically.

Spinal Meningitis

In the majority of cases inflammation of the meninges of the spinal cord is associated with that of the brain. (See "Cerebrospinal Meningitis".) Occasionally, however, the inflammation is limited to the spinal membranes, similar to spinal hemorrhage, being produced by traumatism.

The symptoms of spinal meningitis are practically the same as in spinal hemorrhage, except that the former affection is marked by a sharp rise in temperature at the onset, and by a more progressive character of the symptoms. Absolute recovery is exceptional. The treatment is symptomatic.

Myelitis

This affection is occasionally observed in children principally as a result of traumatism, syphilis and compression of the cord by tuberculous masses and exudates between the dura and vertebræ secondarily to spondylitis. The pathologic process in the cord varies with the etiologic factors. Ordinarily the diseased portion is at first red and soft, and later, yellow, fatty degenerated, atrophied and sclerosed. The lesion may be situated in any part of the cord and accordingly the symptoms differ with the localization. Thus, in disease of the *cervical region* there is first involvement (motor paralysis and sensory disturbances) of the upper extremities, then of the lower, and, if the lesion is very high up, the diaphragm also is affected and respiration is interfered with. In disease of the *dorsal portion* there is paraplegia (with muscular rigidity), with exaggeration of the reflexes, anesthesia of the extremities, paralysis of the bladder and rectum and decubitus. In myelitis of the *lumbosacral* region the paralysis, etc., is the same as in the former lesion, but the muscles are flaccid, show degenerative changes to electric tests, and waste, and the skin and tendon reflexes are abolished. The feet fall into an extended position, so that the instep is on a line with the tibia. In *partial* myelitis the symptoms are less pronounced, extending only to such structures as are innervated by the diseased segment of the cord. In unilateral lesions the symptoms, of course, are limited to the affected side.

The onset may be sudden or slow, according to cause. Acute cases set in with chills, moderate fever, nausea, sometimes vomiting and convulsions, radiating pain in the back and legs, rapidly followed by the aforementioned signs. Cases with gradual onset, *e. g.*, secondarily to spondylitis or compression by extraspinal growths, are manifested by gradually progressing debility of the muscles supplied by the spinal nerves below the compressed area (see p. 168), neuralgic pain, and disturbance of the bladder.

If the primary affection (*e. g.*, tuberculosis, syphilis) can be reached and remedied before destruction of the cord has advanced too far, the progress of the disease can readily be arrested. Otherwise the symptoms continue to grow worse and at best can only be improved by massage, passive motion and faradization, procedures which are generally employed in all forms of chronic paralysis. Attention should be paid to the bladder (catheterization) and bowels, and particularly to the skin, as the tendency to the development of bed sores is very great.

Ataxia Hereditaria (Friedreich), Heredo-ataxie Cerebelleuse (Marie)

This family affection which is traceable through several generations, is of obscure origin. Syphilis in the parents is most probably the cause. The anatomic lesion—degeneration—is situated principally in the cord (the column of Goll, and partly also of Burdach and Clarke) and in some cases also in the cerebellum. The cord as a whole is very thin and small, *i. e.*, arrested in development.

The disease attacks the patient insidiously, between the sixth and fifteenth years of life, with symptoms of simple progressive incoordination of the lower limbs, trunk and arms—irregular swaying resembling that of chorea. Later also nodding of the head. Gradually the tabetic cerebellar gait develops, so that the child is ultimately unable to walk or stand. As the disease progresses, speech becomes peculiar, slightly scanning, heavy and awkward, vision disturbed by nystagmus, and exceptionally by optic atrophy (Argyll-Robertson symptom is absent, while Romberg's is occasionally present), the face expressionless, the general musculature paralyzed, atrophied, the spinal column curved, the feet humpy looking with the toes turned up (*Friedreich's foot*), and, finally intelligence impaired. Unprovoked and uncontrollable laughter is said to be characteristic of the disease. As a rule, sensation and the cutaneous reflexes remain undisturbed; the sphincters are intact until very late, while the tendon reflexes are abolished. The course of the disease is very chronic. The patient is usually bedridden after a period of from five to ten years, but he may continue to live in this state for another ten years.

Disseminated Sclerosis

(Multiple Sclerosis)

The etiology of diffuse and disseminated sclerosis is not definitely known. It is either congenital, and traceable to alcoholism or syphilis in the parents, or is met with in young, apparently healthy and normally developed children some time after traumatism or an attack of an infectious disease.

Its onset is usually insidious with disturbances of motion, loss of memory, and dulness of intellect, soon to be followed by defective speech (at first slow and later scanning), hearing, and vision (nystagmus, amaurosis, and strabismus), spastic paraplegia (weakness and rigidity first of the upper extremities, then of the lower; exaggerated tendon reaction and ankle clonus) and intention tremor. In the later stages of the disease the patient loses control of the bowels and

bladder, suffers from difficult deglutition, attacks of vertigo, loss of consciousness and convulsions, and finally enters into a state of mental and physical exhaustion, paralysis and idiocy. Death occurs after several years.

The symptoms just enumerated do not all prevail in every case. They differ with the location of the sclerosed patches. As a rule, the latter are found not only in the brain but in the medulla and spinal cord as well—chiefly in the white substance.

Treatment.—The disease is very rarely influenced by treatment. Antisyphilitic medication, however, is worth trying.

HEREDITARY PROGRESSIVE MUSCULAR ATROPHIES

(I. SPINAL; II. NEURAL; III. MYOGENIC)

This classification is intended solely to emphasize the principal locations of the underlying lesions. This disease is transmitted from generation to generation and often affects several members of the same family.

I. Spinal Progressive Muscular Atrophy

It is observed in early infancy. It begins with weakness of the muscles of the legs, neck, back, throat, shoulders, arms, hands, fingers and toes. As the disease advances the muscles are completely atrophied (rarely pseudohypertrophied) so that the child is entirely helpless. The reflexes are abolished and the electric reaction greatly disturbed. The disease ends fatally within about four years from involvement of the respiratory muscles and consecutive pneumonia. The lesion consists of atrophy of the cells of the anterior cornu of the entire spinal cord and degeneration of the motor nerve fibers. There is no central involvement, hence, cerebral symptoms are absent. The sphincters are intact. Fibrillar twitching is infrequent.

II. Neural Progressive Muscular Atrophy

(PERONEAL TYPE)

It is characterized by atrophy beginning with the muscles of the legs, especially the peroneal group, and by predominence of sensory disturbances, hyperesthesia or anesthesia. In walking the child lifts the feet high and touches the floor with the tips. If the muscles of the hands are affected, the hands become claw-shaped. Occasionally other muscles are implicated. The patellar and Achilles' tendon reflexes are at first diminished and later abolished. The electric reaction of

the atrophied muscles varies—is normal in some cases, disturbed in others—irrespective of the nature of the atrophy. Fibrillar twitchings are common. The course of the disease is very slow and interrupted by remissions of variable length, and judging by the underlying pathologic anatomy of the affection (degeneration of the respective peripheral nerves, with slight implication of the spinal cord) it is *per se* probably not fatal. Massage, baths and electricity are of benefit.

III. Myogenic Progressive Muscular Atrophy

(DYSTROPHIA MUSCULARIS, PSEUDOHYPERTROPHIC PARALYSIS)

Under this heading are grouped the following four morbid conditions which were formerly looked upon as distinct pathologic entities:

(a) Simple Hereditary Muscular Atrophy. It usually attacks children between eight and ten years of age, and is manifested by weakness and atrophy of the muscles of the back (without pseudohypertrophy), lordosis and paresis.

(b) Infantile Muscular Atrophy (Facioscapulohumeral Type, Landouzy-Déjerine).—As the name indicates it begins in early infancy with atrophy of the face, especially the orbicularis oculorum and oris and the lips. The patient is unable to close the eyes, to point the mouth, and his face becomes expressionless, like a mask. Pseudohypertrophy of the facial muscles sets in later, so also the atrophy of the muscles of the scapulohumeral regions.

(c) Juvenile Muscular Atrophy (Erb).—The atrophy is manifested, at a later age than in the former variety, in the following order: The pectorales, the anterior serrati, the latissimus dorsi, the rhomboidei, and the trapezius muscles, and then the triceps, biceps, brachioradial and brachial muscles. The deltoid is usually strongly hypertrophied.

(d) Pseudohypertrophy (Duchenne).—In this form of the disease the muscles first affected are those of the calves, the extensors of the thighs which become greatly enlarged, and then the long spinal muscles. As the disease progresses the shoulder, arm and lumbar muscles become involved, the deltoid, supra- and infraspinati showing an especial tendency to pseudohypertrophy. The forearm and hands remain free. Owing to the weakness of the erector spinæ and glutei muscles, the patient keeps his trunk thrown backward, "saddleback," and walks with a peculiar waddling gait, with the legs widely separated and the toes barely touching the ground. The gait at times resembles that of bilateral dislocation of the hip. If placed on the floor, the efforts made to rise are very characteristic. Awkwardly

and with difficulty he places first one hand and then the other on the legs, then on the thighs above the knees; and in this manner he "climbs upon himself" until he assumes the erect position (see Figs. 191, 192, 193). In time, the patient becomes unable even to sit up.

The distinction between the different forms of myogenic dystrophia cannot always be made with exactness, as the order with which atrophy begins is not rarely reversed. All varieties of the affection at a late stage present diminution of the tendon and electric reactions, but no reaction of degeneration or central disturbance. Fibrillary

Fig. 191.   Fig. 192.

Figs. 191-193.—Pseudohypertrophic paralysis. Demonstration of mode of rising from the floor by "climbing upon himself."

twitching of the atrophied muscles is absent and local vasomotor disturbances are rare. As the disease advances and the paralyzed muscles contract, various deformities (spinal curvature, talipes, etc.) make their gradual appearance and render the patient totally helpless and bedridden.

The course of the disease is slow, and occasionally interrupted by remissions of variable length, and temporary improvement. Death usually takes place within ten years from the onset of the affection, as a rule, from intercurrent diseases, especially pneumonia.

Treatment.—The treatment in the form of baths, massage, etc., may

prove effective to check the progress of the manifestations, but it is doubtful if it ever leads to permanent recovery.

The disease is attributed to an extraordinary increase of connective and adipose tissues with corresponding atrophy and gradual

Fig. 193.

disappearance of fibers of certain muscles. Slight lesions are not rarely found also in the cord. The etiology is obscure. The absence of fibrillar twitching and of atrophy of the hands and forearms serves as differential points from "Spinal Progressive Muscular Atrophy." (See p. 658.)

Lipodystrophia Progressiva

Lipodystrophia progressiva is a term applied by Simons* to a syndrome beginning most frequently between the fifth and twelfth years, and chiefly affecting females. In this condition, there occurs a grad-

*A. Simons (Zeitschr. f. d. Ges. Neur. w. Psych., Berlin, 1911).

ual, progressive emaciation, beginning in the face, and progressing downward, involving the neck, shoulders, trunk and upper extremities and in most of the cases reported, an increased deposit of fat in the buttocks, thighs and sometimes the legs. The gradual disappearance of fat progresses until the appearance of the face is most characteristic. The cheeks become sunken, the eyes deeply set, the malar eminences prominent, and the temporal regions sunken. When the patient smiles, the cheek is thrown into deep folds and the face generally has a cadaverous appearance. The neck becomes thin, the clavicles and scapulæ extend prominently forward. The intercostal spaces are well marked; the breasts are pendulous and, owing to the disappearance of the fat, hard and nodular.

In contrast to the wasted appearance of the upper extremities and face, the parts below the line of the iliac crests of the individual present a plump appearance; in some of the cases reported even amounting to grotesqueness.

Usually, the attention of the family is first called to this condition by the emaciation which takes place in the face, and the fear of some acute disease prompts them to seek medical advice. The patients themselves complain little or not at all. In advanced cases they sometimes complain of feeling chilly and of excessive perspiration or in other cases, of weakness or nervousness.

In all cases there is a gradual progression of the emaciation of the face, upper extremities and trunk, and increase in size of the lower extremities over a period of ten to twenty years, after which there is spontaneous arrest.

According to I. J. Spear (Archives, Int. Med., January, 1918) this condition is rather uncommon, only 24 cases having thus far been reported.

Treatment.—Therapy seems of no avail. Paraffin injections have been recommended for corrective cosmetic purposes.

Tumors of the Cord and Membranes

Neoplasms of the cord are very rare and, hence, principally of pathologic and diagnostic interest. They may be primary (sometimes congenital) or secondary. Tubercle is the most frequent variety observed; next in frequency are gliomas, syphilomas, lipomas and sarcomas.

The symptomatology depends upon the seat of the growth, essentially resembling that of myelitis, except that it is of gradual development. In benign unilateral tumors the symptoms (motor and sensory paralysis) are limited to the side affected.

Treatment.—Antisyphilitic treatment deserves a full trial, and, if this fails, operative interference should be resorted to.

Peripheral Facial Paralysis

(BELL'S PALSY)

Facial paralysis may be due to trauma, pressure and irritation (swelling or disease) from contiguous structures (glands, teeth, ears) or exposure to cold or draughts.

The symptomatology is essentially alike in all cases irrespective of cause. The paralysis is usually unilateral and effects the muscles of the forehead, the orbicularis oculi and some of the lower facial muscles. As a result, the paralyzed side of the face is lax and expressionless, the

Fig. 194.—Peripheral facial palsy—Bell's palsy. Note inability to close right eye and to frown, as with the muscles of the left side of the forehead. Lower part of face unaffected.

nasolabial fold more or less effaced; the eye remains widely open and the angle of the mouth droops. The paralysis becomes especially pronounced, when the muscles are thrown into action, *e. g.,* on laughing or crying. In severe cases there is also paresis of the soft palate, and impairment of speech and mastication, and occasionally dulness of taste and diminished secretion of saliva. In otic facial palsy there may be disturbance of hearing (deafness; hyperacuteness). In the so-called rheumatic variety or that due to exposure, the onset is usually sudden and accompanied by neuralgic pain. The electric reaction remains normal in mild cases, but is diminished or lost in grave cases.

Prognosis and Treatment.—The prognosis and treatment depend upon the etiologic factors. Traumatic, especially obstetric facial palsy (*q. v.*), where the trauma is slight, usually ends favorably within a few weeks—without any therapeutic measures.

Facial palsy arising from involvement of the facial nerve by aural suppurative processes (middle ear disease; caries of the petrous portion), usually runs a more protracted course, often long after the removal of the cause. Early attention to the ear affection is of vital importance. Cases resulting from dental caries can readily be remedied by treatment, possibly extraction of the diseased tooth.

Rheumatic, grippal, etc., facial palsy ordinarily responds to local heat, the salicylates, quinine and arsenic. Pressure neuritis usually abates with disappearance of the swelling exerting the pressure upon

Fig. 195.—Nuclear facial palsy. Eye muscles are unaffected; paralysis limited to lower part of face.

the nerve. Facial palsy occurring in connection with parotitis calls for no special treatment. Where the pressure is due to a new growth, enucleation of the latter should be promptly undertaken. Recovery is not as rapid in the latter form as in the other varieties.

After abatement of the hyperacute symptoms, a weak galvanic current should be applied four to six times a week, for from two to three minutes at a time. The anode should be held behind the ear, while the different facial nerve branches and muscles are stroked with the cathode.

It has been observed that recovery is assured—after a shorter or longer period of time—in all cases of facial paralysis in which the electric reaction remains normal after a lapse of from one to two weeks. On the other hand, cases which present complete reaction of degeneration of nerve and muscles after that period of time usually offer a doubt-

ful prognosis. Protracted cases may lead to degeneration and shortening of the affected muscles, so that the face appears drawn to the paralyzed side.

Peripheral facial paralysis should not be mistaken for central or nuclear facial palsy. In *cerebral* palsy the muscles of the forehead and eyes, for the most part, escape (*i. e.*, the patient is able to frown and to close the eye on the affected side); the electric reaction is retained; furthermore, the palsy is frequently associated with hemiplegia of the same side. In *nuclear* or basilar paralysis the palsy is usually limited to the lower half of the face (from the mouth down) and is complicated by other symptoms indicating a lesion in the pons, such as cross paralysis and disturbed action of other cranial nerves.

Facial paralysis with lost electric reaction may often be mistaken for the permanent facial paralysis following acute poliomyelitis. (*q. v.*) Indeed, there is reason to believe that the so-called idiopathic form of facial paralysis which resists all methods of treatment is in reality of poliomyelitic origin.

Hemiatrophia Faciei

(Progressive Facial Hemiatrophy)

The nature of this rare affection is still obscure. The pathologic findings point to an interstitial inflammatory process of the trigeminus. It occurs in girls more frequently than in boys, on the left side more than on the right, and exceptionally affects both sides of the face.

It begins with a small part of the face (usually over the fossa canina) turning white, thin, wrinkled, etc. From here the atrophy rapidly spreads to the muscles and bones of the entire half of the face, including the hair. At times the atrophy spreads to the chest and other parts of the body, but finally reaches a permanently quiescent stage. Sometimes there are also anomalies of pigment. It is occasionally associated with scleroderma and exophthalmic goiter. Sensation remains intact and the electric reactions are normal.

Treatment.—The cause of the atrophy being unknown, the treatment must, necessarily, be symptomatic. Paraffine injections have proved very useful to correct the remaining facial deformity.

Polyneuritis

(Multiple Neuritis)

Polyneuritis is an inflammatory, degenerative affection of the peripheral nerves. During the early stage only the sheaths of the nerves

are affected (hyperemic and the seat of minute hemorrhages). As the disease progresses we find connective tissue cells between the nerve sheaths, and red spindle-shaped cells between the nerve fibers, and also parenchymatous changes in the muscles. In severe cases the lesions ascend to the nerve trunks or even to the roots. Its distribution is almost always bilateral and symmetrical. Polyneuritis is very rarely observed in children, since the principal causes of the affection—alcoholism, lead, and arsenic poisoning—are of exceptional occurrence in young children. The most frequent form of polyneuritis encountered is that described as ''Diphtheritic Paresis'' (see Diphtheria), and on very rare occasions it is encountered also in connection with other infectious diseases. In one case under our observation (see Fig. 196) the pain and paresis set in six weeks after an attack of diphtheria.

Fig. 196.—Diphtheritic polyneuritis in a boy four years old, affecting several groups of muscles of the neck, trunk and extremities. Note his inability to rise from the floor.

Fig. 197.—Same case as Fig. 196 two weeks later. Note considerable improvement.

The paresis was very extensive and affected the muscles of the palate and throat (aphonia) neck, trunk, lower extremities and slightly the arms. The four-year-old boy recovered completely within about two months. Strychnine was the only remedy used. The neck was supported by a felt collar.

The onset of multiple neuritis is usually fairly rapid, with numbness, pricking, pain and chilliness of the parts affected. This is followed by the appearance of motor incoordination (ataxia, waddling gait) up to paralysis of symmetrical groups of muscles (e. g., of the hands and feet) or of entire extremities. The symptoms usually get gradually

worse for about four weeks. The lower extremities are ordinarily affected first and the upper later. Genuine foot- and wrist-drop is rare. The same is true of involvement of the muscles of the trunk, and the sphincters. Exceptionally the diaphragm is affected. The motor symptoms are usually associated with sensory disturbances—pain, especially on pressure, along the nerve trunks, hyperesthesia and more rarely anesthesia. The electric and tendon reactions are diminished, and reaction of degeneration is quite common in severe cases.

Fig. 198.—Same case as Fig. 196 six weeks later. He is practically well, except for remaining weakness of the muscles of the neck.

Treatment.—With early treatment—elimination of the poison (sodium iodide, magnesium sulphate, in lead poisoning), mitigation of pain (salicylates, warm baths), tonics (strychnine, iron, etc.), and galvanic electricity and massage,—the prognosis is usually favorable, except when the respiratory muscles are affected. In such cases complicating bronchopneumonia often ends fatally. Occasionally atrophy, with consecutive contractures and deformities, may persist for a long time, and even for life.

	POLYNEURITIS	POLIOMYELITIS	LANDRY'S DISEASE
Onset	Usually slow. Slight fever, if any	Quite acute; often vomiting. Moderate fever	Slight prodromata (pain); no fever
Distribution of paralysis	Symmetrical. Partial. Lower, then upper extremities. Exceptionally other parts of body	Irregular. Complete; often only one limb, or a group of muscles, *e.g.*, shoulder or face.	At first asymmetrical. Ascending. Complete. Legs, trunk, arms, and muscles innervated from the medulla
Hyperesthesia ..	Persistent	Transient	Variable
Anesthesia	Present (partial)	Absent	Absent
Atrophy and deformities	Late and slight	Early	Very late, if at all
Termination ...	As a rule, gradual recovery	Partial, spontaneous, recovery	Usually fatal within two weeks. Exceptionally, recovery

The history of the case is very helpful in the diagnosis. Thus, in multiple neuritis, we are often able to elicit a history of some form of toxemia (infectious disease, especially diphtheria, lead, arsenic, or alcohol poisoning); in poliomyelitis its prevalence in epidemic form may be decisive.

Polyneuritis may occasionally be mistaken for *hereditary ataxia*—very slow in development, involvement of cranial nerves, and mental debility; and *myelitis*—sphincters invariably involved.

B. FUNCTIONAL DISEASES

Spasmophilia

(ECLAMPSIA INFANTUM, TETANISM, TETANY, PSEUDOTETANUS, SPASMUS GLOTTIDIS)

The subject in question is of great clinical interest, and still shrouded in mystery. Spasmodic affections are generally attributed to a number of local bodily irritations which act reflexly upon the central nerve system. We know also that the infantile brain is very vascular, very irritable, very impressionable, lacking in power of resistance and control. We are in the dark, however, as to why the very same etiologic factors are prone to produce mild or severe convulsions in one child and none at all in the other. This apparent discrepancy in action leads one to assume that some children are born with a marked (familial?) tendency to spasmodic affections. This, probably hereditary, spasmodic tendency ("spasmophilia") is distinctly traceable in children of nervous, alcoholic, syphilitic or tuberculous parentage, and exerts its influence principally on the group of functional spasmodic affections presently to be described.

I. Eclampsia Infantum

(Convulsions)

Nonepileptic convulsions are of common occurrence in children, especially in infants under one year of age and are the immediate result of an irritation of the centers in the pontobulbar junction or in the area of Rolando, superinduced either by cerebral anemia or hyperemia. They may occur as a partial, often initial phenomenon of all sorts of acute systemic disturbances, *e. g.*, toxemia from infectious diseases; gastrointestinal intoxication; shock, and trauma; or in consequence of continued reflex irritations, such as phimosis, adenoids, intestinal worms, intense pain from various causes, earache, difficult teething, calculi, and the like. In quite a number of children, a rise of temperature from whatever cause will produce intense convulsions and will continue to recur until the temperature has been reduced. The frequency of the convulsive seizures is within no definite limits—from one attack in several months up to as many as thirty or more attacks in a day. In mild cases the convulsions may be manifested merely by twitching of the lips or eyelids, etc.; in severe cases, however, the convulsions are both tonic and clonic in character. In the beginning, the body is more or less rigid, the head and neck are retracted, the eyeballs are turned upward or roll spasmodically in different directions. The face is distorted and grows cyanotic as breathing becomes labored or temporarily ceases. These tonic spasms are soon replaced by clonic convulsions—irregular and rapid twitching of the extremities and face or of single groups of muscles—which may last from a few seconds to several minutes, may remit, and return with greater violence. With complete cessation of the convulsions the patient usually falls asleep, to wake up apparently free from cerebral disturbance. During the attack consciousness is lost. Occasionally, there may be loss of sensation as well as involuntary urination and defecation, foaming from the mouth and biting of the tongue—a group of symptoms which is generally met in epilepsy. This, together with the fact that eclampsia is not rarely a precursor of genuine epilepsy, should put the physician on his guard in venturing a positive view as to the nature and curability of the spasmodic affection.

Epilepsy differs from infantile eclampsia in that the fit is preceded by an aura, that it is of short duration but nonremittent, and that it is invariably followed by profound sleep—not the light sleep which follows eclampsia. We should bear in mind, however, that these differential signs are much less reliable in epilepsy of children than in adults.

Eclampsia infantum is to be carefully distinguished from uremic convulsions, and spasms accompanying brain disease. In *uremia* there is usually a history (scarlatina?) of suppression of urine. The latter reveals evidences of kidney disease. *Cerebral convulsions* are associated with projectile vomiting, possibly a history of trauma, tuberculosis, otic abscess, and the like. The convulsions of organic brain disease (tumor or abscess) are apt to be more localized and be followed by paralytic phenomena.

Treatment.—When called upon to treat a child in an attack of convulsions, the physician is rarely in position to make exact and scientific discriminations between the different forms of convulsions. It is essential to arrest the convulsions irrespective of cause or effect, since a prolonged attack may end fatally from exhaustion or suffocation. The spasms are best controlled by means of chloroform inhaled from a loosely applied handkerchief, moistened with $\frac{1}{2}$ to 1 teaspoonful of the anesthetic. In this manner the anesthetic may be continued, at long intervals, for hours or days without endangering the life of the patient. As the convulsions subside, we begin to make careful inquiry into their causation and to employ the therapeutic measures indicated in each individual case. Hyperpyrexia calls for hydrotherapy (cold sponge or tub bath); gastroenteric disorders, for emesis (apomorphine 1/16 grain hypodermatically, or ipecac by mouth), catharsis (2 grains of calomel in one dose), and enteroclysis; intestinal worms, for teniafuges (turpentine inhalation, and calomel and santonine by mouth); nervous disturbances, for hot baths with or without mustard, bromide and chloral per rectum or by mouth, and counterirritation in the form of a mustard plaster or mustard-water cloths applied to the spine from the nucha downward. Lumbar puncture is a sovereign remedy in all forms of cerebral irritation associated with increased intracranial or intraspinal pressure and with the usual precautions can safely be employed in convulsions failing to yield to milder procedures.

With cessation of the convulsions due attention should also be paid to the more remote etiologic factors, principally with the view of prophylaxis. The diet should be regulated, the general health improved, rachitis promptly attended to, faulty environment ameliorated, local irritations (*e. g.*, phimosis, adenoids, foreign bodies in the ear or nose, rectal fissures, stomatitis, intense itching, etc.) promptly removed, and all such therapeutic measures instituted as will help to counteract and eradicate the inherent tendency to spasmodic affections.

Occasionally convulsions in children are met which recur for several years irrespective of all prophylactic and therapeutic meas-

ures, and then suddenly cease. In such cases the cause will probably be found in some obscure disturbance of the endocrine glands, giving rise to some form of autointoxication.

I:	Natrii Bromidi	3 i	4.00
	Antipyrinæ	3 ss	2.00
	Tr. Ammonii Valerianatis	3 ii	8.00
	Syr. Lactucarii	3 iv	15.00
	Aq. Aurantii Flor.	q. s. ad f 3 ii	60.00
	M.		

S.—One teaspoonful every three to six hours, for a child two years old. (General nerve sedative.)

II. Tetanism

This term is intended to denote a peculiar form of continued muscular hypertonicity occasionally observed in very young infants with markedly lowered vitality, be it as a result of prematurity, syphilis or

Fig. 199.—Tetanism during acme of spasm. Note characteristic position of the extremities.

Fig. 200.—Tetanism. Same case as Fig. 199 during partial relaxation of spasm.

chronic gastroenteritis. The onset of the spasticity is fairly rapid, and in severe cases, when fully established, the posture assumed by the patient is pathognomonic (Fig. 199). The head is moderately retracted, the facial muscles are contracted, the jaws are firmly set together, the forearms are flexed upon the arms and the hands are tightly clenched, so as to form firmly closed fists. As a rule, the legs are bent angularly and the feet either overlap each other or are arched. The muscular contractures relax off and on (Fig. 200), more especially during profound sleep, but never subside entirely. The hypertonicity increases on handling the baby, but it never interferes with feeding. With improvement of the general health of the baby, the contractures disappear.

As may be noted from the accompanying illustrations, tetanism is a typical clinical picture easily to be differentiated from similar spasmodic affections. On the first examination of the patient we may sus-

Fig. 201.—Same case as Fig. 199 three months later.

pect either tetany, tetanus, or eclampsia, but on careful analysis of the symptomatology of the affections, the erroneous impression can readily be dispelled. Tetanism differs from *tetany* by its more gradual development and almost continuous persistence for several months; any kind of handling of the baby increases its muscular hypertonicity, while in tetany the attacks may be brought about or aggravated only by pressure upon large trunks of nerves or arteries (Trousseau's phenomenon), electric excitability (Erb's phenomenon), or irritation of the facial nerve (Chvostek's sign). *Tetanus* is an acute disease, preceded by an infection, as a rule accompanied by difficult deglutition and respiratory embarrassment and usually ending fatally within a week. *Eclampsia infantum* occurs in attacks and is associated with loss of consciousness. In the same manner we can promptly exclude

so-called *meningismus;* moreover, none of these spasmodic affections of infancy ever give rise to the characteristic contractures of the extremities just described.

With improvement in the general condition, the spasticity gradually (within a week or a month or longer) subsides. Few babies in that dilapidated state survive, however, the persistent gastroenteritis and increasing exhaustion.

Treatment.—Prophylaxis and therapy the same as in tetany (q. v.) except that there is no indication for the employment of hypnotics.

III. Tetany

This disease is characterized by intermittent, somewhat painful, contraction of certain groups of muscles, especially of the extremities, with exaggeration of the mechanical and electric irritability. The spasm is bilateral and usually sets in abruptly without loss of consciousness. The hands assume a very peculiar shape greatly resembling that of holding a pen or of making a strenuous effort to restrain a spirited horse. Thus, the arms are pressed against the chest, the hands are bent on the forearms, the fingers are flexed upon the palms, the phalanges are extended, the thumbs are turned inward, so as to be covered by the other fingers, and the wrists are flexed in pronation. When the lower extremities are affected, the legs are adducted and the plantar surfaces of the feet are strongly arched, with a tendency to an equinovarus position. Occasionally the tetanic spasm extends to the neck and back, and exceptionally also to the laryngeal and other muscles of the body. On the other hand, cases of tetany are encountered in which the spasms are entirely wanting or barely indicated. These "latent" or passive forms of tetany may frequently be brought into activity by energetic pressure upon the main trunks of the nerves or vessels (*e. g.,* bend of elbow). This peculiar mechanical manifestation is spoken of as "Trousseau's phenomenon," and forms one of the three positive signs of tetany—the so-called "triad of tetany." The other two signs of tetany are those of Chvostek and Erb. "Chvostek's phenomenon" is based upon exaggeration of the mechanical irritability of the motor nerves, especially of the face (facialis phenomenon), and consists of lightning-like contractions of the face superinduced by percussion (with the finger or hammer) over a branch of the facial nerve while the face is in a state of perfect rest. "Erb's phenomenon" is based upon electric excitability of the motor nerves and muscles, and Escherich and von Pirquet maintain that we have not only a reaction or muscular response to local cathodal opening and closure, but that a current of 4 ma. is sufficient to produce muscular

contraction or anodal closure and opening as well "anodal suscepti-
bility."

The duration of the tetanic attack varies from a few minutes to
several hours or longer. When they have lasted some time there usually
develops edema of the dorsi of the hands and feet. The spasms may
recur once or several times daily or but once in several days. In the
great majority of cases the disease usually subsides within a few days
or a month or two, without any permanent sequelæ, provided suitable
treatment is instituted early. Zonular cataract may occasionally form
a sequel of tetany and is probably due to the effects of faulty metabolism.

Fig. 202.—Tetany in a child eleven months old. Note characteristic attitude of
hands and feet.

Whether or not the immediate cause rests upon functional or or-
ganic disturbance of the thyroid glands or parathyroids (hemorrhage
in the epithelial bodies) is still subject to great differences of opinion.
Escherich and his pupils strongly favor this theory and endeavor to
prove that the faulty distribution of calcium was due to interference

with the functions of the parathyroid. Howland and Marriott* insist that enough studies have been made to show that parathyroid lesions in infancy are the exception and not the rule. Furthermore, they maintain that parathyroid lesions as severe as have been found after tetany may occur in patients who, during life, have shown absolutely no evidences of this symptom. Their conception of tetany is that some factor, at present unknown, causes a reduction of the calcium content of the blood. When this drops to an amount roughly between 6 and 7 mg. of calcium per hundred c.c. of serum, frank evidences of tetany arise. This amount, however, is not the same with all individuals. With some it may be as low as 5.5 or 6, with others as high as 7.5. These symptoms occur in outbursts so long as the calcium remains low. When the calcium rises, the symptoms disappear. The height to which the calcium must rise in order that the symptoms must disappear is also somewhat variable. Wilson[1] and his co-workers at the Johns Hopkins Medical School found that, following parathyroidectomy in dogs, the equilibrium between acids and bases is displaced in favor of the bases, and that in tetany developing after such a procedure there is well marked alkalosis. The results have been confirmed by McCann[2] at the Harvard Medical School, who agrees that there is a marked increase in the carbon dioxid-combining power of the blood plasma, coincident with the development of tetany.

Treatment.—The treatment, especially with the view of prophylaxis, is essentially the same as employed in rachitis—corresponding to the apparent relationship that exists between the pathogenesis of rickets and that of tetany. Similar to rickets, tetany occurs in infants chiefly of a half to two years of age. Like rickets tetany shows a predilection for poorly fed and poorly housed children, and, finally, as in rickets, the immediate cause of tetany seems to be some form of intoxication, intestinal or otherwise.

The diet should be regulated, as to quality and quantity. Young infants should, if possible, receive breast milk. The intestinal tract should be cleansed with calomel by mouth, lavage and low enemas. For the relief of severe contractions prolonged warm baths, bromides and chloral will usually prove efficient (see "Rachitis"); and in view of the fact that there is an insufficiency of calcium in the blood and also that in surgical parathyreopriva calcium is found to arrest the tetanic spasm, we are fully justi-

*Howland, John. and Marriott, W. McK.; Observations on the Calcium Content of the Blood in Infantile Tetany and on the Effect of Treatment with Calcium. (Bull. Johns Hoskins Hosp., Vol. xxiv, p. 235, 1918).
[1]Wilson, D. W.; Stearns, Thornton, and Janney, J. H., Jr.: J. Biol. Chem. 21, 169, 1915; Wilson, D. W.; Stearns, Thornton, and Thurlow, M. D.: Ibid. 23, 89, 1915.
[2]McCann, W. S.: A Study of the Carbon Dioxide-Combining Power of the Blood Plasma in Experimental Tetany, J. Biol. Chem. 35, 553 (Sept.) 1918.

fied in favoring its administration also in infantile tetany. Syr. calcii lactophosphatis ($\frac{1}{2}$ dram) or calcium lactatis (2 grains) three times daily are useful preparations. Phosphorus with cod liver oil should be given a fair trial.

Late or Puerile Tetany is met with in children over three years, and is manifested chiefly by carpopedal spasm of brief duration.

IV. Pseudotetanus* (Escherich)

This affection differs from *tetanus* principally by its predilection for the muscles of the trunk, and its afebrile course; from *tetany* by its spasticity being continuous, and from *tetanism* by the fact that it attacks children of from four to fourteen years of age (instead of infants) who are apparently enjoying perfect health. The pathogenesis of the disease is still unknown.

The patients (usually boys) suddenly complain of stiffness in the legs and inability to walk about. The rigidity rapidly extends to

Fig. 203.—Pseudotetanus. (After Pfaundler and Schlossmann.)

the back and head, so that the patient lies motionless like a log, except for his ability to make free use of his arms and hands. The affected muscles are maximally contracted, prominent, and as hard as marble. The facial muscles except those of the eyes also are in a state of tonic spasm, so that the facial expression is that of trismus, the teeth are firmly set together and barely separable with force. Nevertheless, there is but little difficulty in feeding the patient. The rigidity is in partial abeyance during sleep as well as during perfect rest, but greatly increased—up to painful opisthotonos, spasm of the diaphragm, etc.—by all sorts of bodily or mental irritations. During the height of the disease such spasmodic paroxysms may occur also spontaneously several times a day and are usually followed by profuse sweating.

*There is considerable diversity of opinion regarding the existence of such a clinical entity. It surely is of very rare occurrence.

The spasmodic condition persists without apparent variation for from three to six weeks, whereupon the contractures gradually (within from two to four weeks) abate never to return.

Treatment.—The treatment is symptomatic. (See "Tetany".) Gavage, if necessary.

V. Spasmus Glottidis

(LARYNGOSPASM)

Spasm of the glottis is a disease of infants of from six to twenty-four months old, the age in which rickets is most apt to prevail. It is closely related to and a frequent partial phenomenon of tetany (usually shows Trousseau's and Erb's signs) and seems also to rest upon the identical pathogenesis of the latter disorder.

The spasmodic attack is manifested by sudden deep inspiration, dyspnea, apnea, pallor and later cyanosis of the face, fixation or rolling of the eyes, and more or less marked rigidity of the body. At the end of a few seconds breathing is resumed after a noisy expiration. In severe cases the spasm not rarely extends to the diaphragm and to the entire musculature of the body.

The attacks usually recur at shorter or longer intervals (several times a day!), and, if not terminating fatally, which may occasionally take place very suddenly even during a simple attack as a result of asphyxia, they gradually subside after a few weeks or months. In mild cases recovery is the rule. The physician should be guarded, however, in the prognosis.

Spasmus glottidis can readily be distinguished from other forms of laryngeal stenosis (*e. g.*, retropharyngeal abscess) by its intermittency and noiselessness, between each attack. It should not be confounded with the momentary apnea ("holding the breath"), frequently observed in children during a fit of crying. (See "Congenital Stridor" and "Thymus Hypertrophy".)

Treatment.—As the physician rarely has the opportunity to witness an attack of laryngospasm, his efforts must be directed chiefly toward its prevention. This is best accomplished by antirachitic treatment (*q. v.*), including calcium, careful attention to the alimentary tract, and calming of the irritability by means of small doses of sodium bromide. (See "Eclampsia".) Severe attacks call for stronger hypnotics.

A severe attack may sometimes be aborted by dashing cold water in the child's face, exciting choking motions by pressure upon the root of the tongue, and exciting sneezing by irritating the nasal mucous membrane. In some cases light ethyl chloride or ethyl bromide anesthesia

may be tried. Timely intubation and artificial respiration have saved some babies from immediate death.

Chorea Vera

(St. Vitus's Dance, Chorea Minor, Sydenham's Chorea)

Genuine chorea is an acute, infectious, sporadic and epidemic affection characterized by spontaneous, irregular movements of the voluntary musculature, and by a special tendency toward cardiac complications.

The specific causal microorganism of this disease is still unknown, but is probably closely related to that of rheumatic affections, with which chorea is occasionally associated. Other infectious diseases (such as exanthemata), fright and mental overwork serve as predisposing causes.

The onset of chorea is preceded by prodromata varying in duration from a few hours to a few days. They consist of fretfulness, fatigue, pain in the extremities, restless sleep and occasional twitching. After the prodromic stage the actual attack may be precipitated abruptly and with full force, or come on gradually and run a mild course. The cardinal symptoms of the disease are irregular, awkward, involuntary, muscular movements—hasty and beyond control—which cease only during sound sleep. The movements intermittently involve various sets of muscles never letting up a moment while the patient is awake. The movements are intensified when the patient is conscious of being observed, and tries to control them, or attempts to perform some voluntary action. The shoulders, one or both, jerk upward or downward, the arms rotate from side to side, or are forcibly thrown backward, the hands are engaged in incomplete extension, flexion, pronation or supination, while the fingers are bent, extended or shoved one over the other so that the patient is unable to hold an object firmly, to write, to button a garment, etc. The head sways from side to side, often describing a semicircle, or is dropped downward so that the chin touches the chest wall. The facial muscles twitch, and produce grotesque distortions of the face and mouth. The forehead is wrinkled, the eyes open and close, the patient seeming to cry or laugh. In one case under our observation the iris (!) was involved so that the pupils contracted and dilated almost incessantly. The tongue participates in the movements, causing difficulty in eating and drinking, and defective speech up to aphasia. The movements of the lower extremities vary with the intensity of the attack, in severe cases be-

ing of such nature that the patient is unable to stand, sit or lie still, and frequently falls, stumbles, or is thrown out of bed and injured. During the acme of the attack it is not uncommon to find irregular respiration and arrhythmia of the pulse—both from implication of the respiratory muscles and the heart (*chorea cordis*). However, notwithstanding the intensity of the movements the patients rarely complain of being fatigued, in fact a great many children seem otherwise in perfect health. The temperature is normal, the digestion good, sensory disturbances are usually rare and slight (hyperesthesia along the course of the nerve trunks), the patellar reflex is somewhat exaggerated, but the cutaneous sensibility and reflexes are unaltered.

If left untreated the active stage of the disease lasts from four to six weeks; then the symptoms gradually diminish and may disappear entirely a few weeks later. Some cases run a mild course from beginning to the end, at no time presenting the aforementioned grotesque muscular excursions. This is especially prone to occur if treatment is begun early, and persisted in.

The intensity of the attack stands in no relation to its duration; on the contrary, cases of slow development and moderate severity may run a chronic course and suffer relapses, while violent cases often respond to a few weeks' treatment. This incongruity is often observed also as regards complications; mild cases being not rarely associated with fever, inflammation of the joints, pleura, pericardium or endocardium, whereas severe chorea may run its course without any untoward results. In reference to heart complications which is supposed to occur in about 20 per cent of cases, it is well to remember that not every blowing heart sound heard in chorea is indicative of valvular lesion; the majority of these adventitious sounds, especially those heard at the base, disappear, perhaps, never to return. On the other hand, heart lesions have been found at the autopsy without any indications of their presence during life, a fact which strongly emphasizes the necessity of prophylactic measures (perfect rest) being taken against heart disease during the active stage of the disease.

Sometimes the muscular disturbance is limited to one-half of the body (*hemichorea*), showing that the lesion is localized in one hemisphere of the brain. This form of chorea is more serious than bilateral chorea. It is often associated with paresis of the extremities, one or both (*chorea paralytica; chorea mollis*), and changes in the psychical condition, *e. g.*, melancholy, hallucinations.

Notwithstanding the grave nature of the affection, the prognosis of chorea, on the whole, is favorable. A fatal termination is exceptional.

This may occur either as a result of complicating heart disease, or from some, as yet unknown, effect upon the central nerve system. To the latter class belong the cases associated with delirium and prostration (*chorea insaniens*). On the other hand, the prognosis as to permanent recovery is not quite promising. Recurrences are frequent, and as previously mentioned, the tendency to permanent heart disease is great.

Treatment.—With these facts in view, the urgency of instituting preventive measures against chorea is obvious. This is strongly emphasized by the observation that chorea may appear in epidemic form (it is quite common to find several members of one family to be attacked simultaneously or within a brief period of time). I am not referring to the hysterical "pseudochorea" not rarely encountered in epidemic form in girls' boarding schools. (See "Hysteria".) Prophylaxis is best accomplished by isolation of the patient. This is imperative in hospitals, asylums or private schools where several inmates are congregated in close quarters. Girls, between six and twelve years of age particularly, should be kept apart, as they are very susceptible to chorea: about 70 per cent of the cases are met in girls, probably because of their poorly developed body musculature. In recurrent chorea the teeth, tonsils and adenoids should be looked after.

The active treatment consists principally of perfect rest in bed in an airy and sunny room, and avoidance of all mental excitement. While the choreic movements are very pronounced, the patient should be kept in a well-padded bed (to avoid injury) day and night, but, as the symptoms improve, she may be allowed to sit up or be around and about for a few hours at a time. A warm bath with a cool sponge once or twice a day and a daily colon flushing are very salubrious. The food should be bland, nutritious, and preferably liquid or semi-solid (milk, cereals, broths, fruit juice, etc.), especially when mastication and degulutition are difficult. Arsenic in the form of Fowler's solution is the remedy *par excellence* in all cases of chorea, except when associated with marked paresis. It should be begun with in ½ drop doses for every year of the child's age, and increased by ½ a drop every other day. Should the urine show the presence of albumin, the lids become puffy, the stomach irritated (pain or nausea), it is advisable to go back to the original dose, or to discontinue it entirely for a few days. In the so-called paralytic cases the cacodylates of arsenic, strychnine and glycerophosphates, administered either by mouth or, preferably, hypodermically, often act exceedingly well. Whenever sore throat or rheumatic pain has preceded the attack of chorea, the salicylates, with or without digitalis, should be pushed to full tolerance. During the acme of the disease, the bromides, and

more powerful hypnotics, if needed, will be found to act kindly in re-
ducing the severity of the choreic movements, allaying the nerve irri-
tability and inducing sleep—all of which being essential to the recovery
and maintenance of the strength of the patient. In very grave cases
chloroform anesthesia may very cautiously be resorted to. Many
years ago I reported a number of cases of protracted chorea which
were greatly benefited by lumbar puncture. The suggestion recently
made to inject an autogenous blood serum into the spinal canal has
failed to meet with favor. The same holds true for the intraspinal
injection of magnesium sulphate. Finally it is well to bear in mind
that a number of cases will run their course, uninfluenced by any
method of treatment, and possibly be harmed by overtreatment. In
refractory cases we may try a milk- and meat-free diet, for a week
or two.

> ℞ Liq. Potassii Arsenitis
>   Aq. Aurantii Flor.   āā ʒ ii │ 8.00
>   M.
>   S.—Begin with one drop for every year of the
>   child's age and increase by one drop every other
>   day, up to full tolerance. To be well diluted in
>   water.

> ℞ Natrii Salicyl
>   Natrii Bromidi   āā ʒ i ss │   6.00
>   Mist. Rhei et Sodæ   ʒ iv   │ 15.00
>   Aq. Destil.   q. s. ad f ℥ ii │ 60.00
>   M.
>   S.—One teaspoonful every four to six hours,
>   for a child six years old.

> ℞ Ferri Sulph. Exs.   gr. x │ 0.60
>   Pulv. Chocolate   ʒ i │ 4.00
>   M. Div. in pulv. no. xx.
>   S.—One powder after each meal.

Habit Spasm

(Tic)

Children of a nervous temperament quite frequently acquire the
habit of spasmodically moving the head (swaying, rolling or nod-
ding), face (*tic*), fingers and hands, which, if not immediately stopped
by strict discipline, is apt to persist for weeks and months. In some
cases the bad habit is traceable to faulty wearing apparel. For ex-
ample, head nodding in girls from poorly fitting hats, head swaying
in boys from a too tightly fitting collar, etc. Habit spasm should not
be confounded with chorea.

A similar spasmodic condition has been described by Henoch as *"chorea electrica."* It occurs in children from nine to fifteen years old, in the form of lightning-like spasms, especially of the neck and shoulders, as though produced by a galvanic current. This spasm seems to be identical with *"paramyoclonus multiplex"* (a neurosis marked by shock-like muscular contractions, which are bilateral and do not, as a rule, affect the hands and face) but may be hysterical in nature. Electricity does well in these cases, probably by suggestion.

Spasmus Nutans

(Spasmus Rotatorius, Head Nodding)

The disease in question is of obscure origin. It is usually seen in infants of from four to eighteen months of age, chiefly in those suffering from rachitis. The spasmodic movements are generally limited to the muscles innervated by the cervical plexus and the accessory nerve, notably the recti capitis, longus colli, scaleni and sternocleidomastoid. In consequence of the irritation, the head rotates from side to side or shakes anteroposteriorly at a variably rapid (every second) pace, with occasional interruption, but ceases entirely only during sleep or temporarily while blindfolded (Caillé). The head nodding is usually associated with nystagmus and more rarely strabismus or rolling of the eyeballs. In some cases some etiologic relation seems to exist between spasmus nutans and visual disturbance, but whether the defect be in the muscle or nerve supply is still a matter of conjecture. Henoch attributes the association of the nystagmus with the rotatory movements of the head to the close proximity of and extension of irritation from the ocular nuclei to the nuclei of the nerves and muscles which rotate the head.

The spasmodic movements gradually disappear in the course of a few weeks or months, after improvement in the general health. (See Rachitis.)

Spasmus nutans may be confounded with "juvenile congenital nystagmus" (associated with marked visual defects, *e. g.*, disease of the retina, lens, etc.) ; with brain disease which can readily be recognized by the concomitant symptoms, and with "epilepsia nutans" (*q. v.*).

Hysteria

Hysteria is a neuropsychosis, a product of faulty environment and education.* It is rare in children under eight years of age, but quite common in older ones, especially in girls.

---

*Sheffield, H. B.: A Contribution to the Study of Hysteria in Children. (New York Med. Jour., September 17 and 24, 1898.)

The onset of hysteria can frequently be traced to some sudden violent emotion (shock) with, or more rarely, without bodily injury. The attendant circumstances at the time of the psychic disturbance often serve to determine the seat of the hysterical lesion, *e. g.*, hysterical deafness after an explosion, paralysis or contracture of an extremity, after a trivial injury.

The symptomatology of hysteria is characteristic for its multiplicity and mutability. It may closely simulate that of any organic disease, but its spuriousness can usually be detected after careful scrutiny. The diagnostic perplexities augment, however, with accidental concurrence of some acute affection or preexistence of a chronic organic disease.

Paralysis of the extremities with or without contracture forms a frequent hysterical manifestation. It may appear in the form of paraplegia, monoplegia, or hemiplegia, and thus resemble myelitis, poliomyelitis, or cerebral paralysis. In hysterical "spinal" paralysis, however, there is rarely absolute loss of muscular power. Muscular atrophy is absent or slight, and electric irritability remains normal. In hysterical "cerebral" paralysis, also, the loss of power is rarely complete and the leg is often more affected than the arm. The face usually remains uninvolved. A peculiar form of either continuous or intermittent pseudoparalysis is occasionally met with in children, which has been described by Blocq as "astasia-abasia." In this condition the muscles of the lower extremities can be freely used except in standing or walking. If the latter is attempted, the patient immediately falls to the ground or begins to tremble and topples over, or manifests ataxic sympoms (cerebellar type). The difficulty in walking is sometimes overcome after a few steps are taken.

The hysterical contractures may involve the articulations, groups of muscles or a part of a muscle. As a rule, the joints of the tapering extremities are most frequently affected. All sorts of deformities may arise which may greatly resemble genuine joint and bone disease (*e. g.*, hip-joint disease, spondylitis, talipes, etc.) and lead to errors in the diagnosis. The more sudden onset, the irregularity of its course, the tendency to change its situation and the concomitance of other evidences of hysteria, all help the exclusion of organic disease. At a later stage the diagnosis of hysterical contracture can frequently be made by the absence of local thickening, or active inflammation of the bone or muscle and its disappearance under anesthesia. Where part of a muscle is affected the contracture may give rise to circumscribed swellings. Allied in nature are also the so-called "phantom tumors" occasionally observed on the lower portion of the abdomen,

and the peculiar "ballooning" of the hypogastrium manifested with each expiration. Occasionally the abdominal enlargement is general and not rarely accompanied by local tenderness. Furthermore, the hysterical tympanites may be associated with vomiting, anorexia, singultus, disturbed respiration, retention of urine, etc., and thus give rise to the clinical picture of peritonitis, which may test the skill of even the best diagnostician. As a rule, obstipation and fever are ab-

Fig. 204.—Hysterical phantom tumor of the abdomen. (After Pfaundler and Schlossmann.)

sent in these cases and the vomiting is not so persistent as in true peritonitis. Of course, vomiting, anorexia, tachypnea, etc., may exist independently of the hysteria and greatly obscure the diagnosis.

The symptoms thus far enumerated represent principally the neurotic element of hysteria. To those may be added the occasionally

occurring cataleptic states, spasms of the laryngeal muscles (croup), dysphagia, aphasia, aphonia, with spells of coughing, singing, or stuttering, asthma, amblyopia, hemianopsia, contraction of the visual field, amaurosis, and blepharospasm.

In another group of cases the psychic element predominates. Here, too, however, there is generally a great display of spasmodic and convulsive movements ranging between simple or choreic tremor to marked epileptiform convulsions (hysteroepilepsy). The movements may assume the form of athletic exercises, such as rowing, swimming, punching, etc.—*chorea rhythmica;* or the patient may act as though possessed, climb walls, turn somersaults, and perform all sorts of stunts —*chorea magna.* Still more advanced cases of hysteria may be manifested by attacks of sopor, night terrors, somnambulism, hallucinations, delirium and mania. Hammond, in his treatise on "Spiritualism" (1876), refers to several journalistic reports of epidemics of hysteria as they occurred in this country two centuries and more ago. One of the first documents of this kind appeared in a New England paper, in 1688, and reads as follows:

Four children of John Goodwin, of Boston, remarkable for their piety, honesty, and industry, were in the year 1688 made the subject of witchcraft. The eldest, a girl about thirteen years old, had a dispute, about some linen that was missing, with a laundress whose mother, a scandalous Irishwoman of the neighborhood, applied some abusive language to the child. The latter was at once taken with "odd" fits which carried in them something diabolical. Soon afterward the other children, a girl and two boys, became similarly affected. Sometimes they were deaf, sometimes they were blind, sometimes dumb, and sometimes all of these. Their tongues would be drawn down their throats, and then pulled out upon their chins to a prodigious length. Their mouths were often open to such an extent that their jaws were distorted and were then suddenly closed with a snap like that of a spring lock. The like took place with their shoulders, elbows, wrists and other joints. They would lie in a benumbed condition and be drawn together like those tied neck and heels, and presently be stretched out, and then be drawn back enormously. They made piteous outcries that they were cut with knives, and struck with blows, and the plain prints of wounds were seen upon them. At times their necks were rendered so limber that the bones could not be felt, and again they were so stiff that they could not be bent by any degree of force.

The next authentic account is offered by Rev. Dr. Davidson.* While relating the proceedings of a Kentucky camp meeting, in the year 1800, the writer remarks that "small children had taken part in the religious ceremonies, which consisted in part in the following feats: Simple jerking of the arms from the elbow downward. The head was thrown backward with a celerity that alarmed spectators, causing the hair, if it was long, to crack and snap like the lash of a whip. The children would bounce from place to place like a foot ball, or hop around with head, limbs and trunk,

*History of the Presbyterian Church in Kentucky.

twitching and jolting in every direction. Sometimes the head would be twitched right and left to a half round with such velocity that not a feature could be discerned.''

This hysterical method of worshiping seems to have been ''contagious'' in character, for about the same time several such epidemics are recorded, foremost of which is that reported by Rev. John Wilkinson,* who realized the morbid basis of the religious ceremonies. It may be noted here that this contribution on hysteria seems to be the first one ever published in an American medical paper. ''This disease,'' the divine begins, ''made its appearance early in the summer of 1803, and increased in its effects with astonishing rapidity until the latter end of that season. I have known some persons as young as six or seven years of age, and others, I think, upward of sixty affected * * * There is scarcely one girl in ten between the age of ten to twenty that has not had or now has the exercise * * * The paroxysms continued from a half an hour to an hour and upward. The agitation consisted in twitching, retching, groaning, jerking and laughing. Premonitory symptoms were compression or weight in the chest or about the heart. The motion gave relief. No other complaints of corporeal pains were made.

They all agree in asserting that during these exercises the senses remain in full vigor, and that even in their silent exercises they know everything that is passing about them. They also say that their mental faculties during the paroxysms are preternaturally active and strong * * * When a person is in the silent exercise, if a pin or a needle be introduced through the skin, it will cause no emotion or complaint, but will produce the sensation of pain.''

Epidemics of this kind occurred also in 1835, 1846, and 1870, but for the sake of brevity we will omit their full discussion.

Hysteroepilepsy is comparatively rare in children. An attack is usually preceded by emotional excitement, globus hystericus, etc., and may be induced by pressure upon sensitive areas—hysterogenic zones —of the body, such as the hypochondriac or spinal regions. Hysteroepilepsy differs from genuine epilepsy in the following respects:

Epilepsy	Hysteroepilepsy
Onset sudden	Preceded by emotional excitement
Consciousness entirely lost	Partially preserved
Convulsions preeminently clonic	Tumultuous, accompanied by moaning, screaming, crying, etc.
Duration short, followed by stupor	Longer; followed by restlessness

Hysteria generally proceeds a very chronic course, with temporary improvement and relapses. Of course, it very much depends upon the etiologic factors, the time when treatment is begun and the energy with which it is carried out.

Without denying the transmissibility from parent to offspring of a certain degree of nerve instability which may predispose to hysteria,

*Philadelphia Med. and Phys. Journal, pp. 87-96, 1805.

in the great majority of instances this disease is acquired as a result of harmful influences of faulty environment and education. A child repeatedly seeing its mother, for example, in a state of emotional excitement or frenzy, sooner or later, consciously and deliberately, or otherwise, learns to imitate its mother's hysterical performances, the habit of imitation gradually leading to aberration of the normal cerebral functions. Unable as the mother is to control her own abnormal actions and feelings, she can hardly be equal to the occasion to guide her children in the right direction. On the contrary, the child is allowed to have its own way, is made the central figure of the household and spoiled by overtenderness. If, in addition, such methods of education are adopted as will overtax the child's mental capacity (*e. g.*, the study of music, painting, emotional recitations, etc., in addition to arduous school work), a deranged state of mind sooner or later supervenes which is most susceptible to the aforementioned pernicious influences. Less potent in the predisposition to hysteria are the use of alcoholic beverages, acute infectious diseases, prolonged disturbance of sexual (masturbation!), digestive and circulatory (anemia) systems, in fact, anything that will undermine the physical or mental condition of the child.

Treatment.—With these principal etiologic facts in view, the indications for the treatment of hysteria in children are self-evident. The patient should be removed from the hysterical environments, and placed under the care of one who with kindness but firmness can control his destiny. Change of residence from the noisy city to the restful country often works wonders. The child should lead an outdoor life, and every effort should be made to raise his general bodily development. The food should be ample and nutritious, free from alcoholic beverages. Milk foods should be given preference to meats. The education should be restricted to the simplest school work, or, for a time at least, entirely suspended.

The active treatment of hysteria is essentially symptomatic. Warm baths and cold showers and general massage are useful in all cases. Paralysis and contractures frequently yield to electricity, its action being probably suggestive in nature. Suggestion by electricity or other spectacular procedures are also effective in relieving local conditions, such as aphonia, stuttering, blindness, and the like. Hystero-epilepsy and maniacal outbreaks call for isolation, rest in bed, and the administration of small doses of the bromides and valerian. Disregard of the patient's complaints and severity will often cure some hysterical phenomena where kinder therapeutic measures ordinarily fail.

℞	Natrii Bromidi	ℨ i	4.00
	Ext. Humuli Fl.	ℨ iij	12.00
	Infusi Valerianæ Rad.		
	Aq. Aurantii Flor.	āā ℥ j	30.00
	M.		

S.—One teaspoonful every four hours, for a child ten years old.

Dystonia Musculorum Deformans

(Progressive Torsion Spasm of Childhood)

At a meeting of the Berlin Psychiatric Society, December 17, 1910, Ziehen[1] demonstrated a child ten years of age with a spasmodic affec-

Fig. 205.—Progressive torsion spasm. (J. Ramsey Hunt.)

tion of unusual type. Four similar cases had come under his observation, three in members of the same family. In all, the affection had been gradually progressive and was characterized by spasm and hypertonicity of the musculature, with curious twisting and torsion movements of the extremities and trunk. The muscular spasm was considerably increased by active movement and diminished during

[1]Ziehen: Tonic Torsions (Neurol. Centralbl., xxx, 1909; Allg. Ztschr. f. Psychiat. 1911, lxviii, 281.)

rest. The gait and station were chiefly affected and there was marked lordosis of the spine. The tendon reflexes were present but difficult to elicit, because of the tension and torsion of the muscles. Sensation and intelligence were not affected, and, in one of the cases, necropsy had revealed no essential lesion. All the patients were Russian Jews.

Since then numerous cases of this affection have been reported by J. Ramsey Hunt,* who made a special study of the subject in this country.

*Jour. A. M. A., Nov. 11, 1916.

AMENTIA

IDIOCY AND THE ALLIED MENTAL DEFICIENCIES

I. In Infancy and Early Childhood

NATURE AND PATHOGENESIS

Amentia is not an affection *sui generis,* a precise morbid entity, but merely a syndrome of a large group of congenital and acquired pathologic conditions, principally of the brain and the ductless glands. The degree of mental debility is very variable and not rarely incommensurate with the extent and gravity of the causal organic lesion. Thus, profound idiocy is frequently encountered with seemingly insignificant structural changes in the brain or elsewhere, and *vice versa,* gross brain lesions may occasionally be accompanied by only slight feeblemindedness. As a rule, however, definite postmortem findings, with predominance of characteristic lesions in certain types of cases are observed in the great majority of cases of amentia, so much so, as to permit—in accord with the underlying pathologic anatomy—to classify idiocy and the allied mental deficiencies into distinct groups (*e. g.,* idiocy with microcephalus, hydrocephalus, athyrosis, etc.), which will presently receive due consideration.

In almost all forms of amentia the cerebral convolutions are more or less modified and irregular in outline and diminished in number. They are either agglutinated or separated by widely gaping grooves. Frequently there is an appreciable difference in the size, shape and weight of the hemispheres, or an asymmetry of the corpora striata, the peduncles, or the pyramids, or even the absence of one or more of these bodies. Similar changes are often observed in the cerebellum, and occasionally in the pons, medulla and spinal cord.

Microscopically, we can readily detect an arrest of development or disease of the nerve cells of the brain cortex, of the nerve fibers, and of the neuroglia. The nerve cells are immature, irregularly arranged or numerically deficient. The nerve fibers are greatly diminished in number, more especially in the frontal and parietal lobes, which seem most concerned with the highest intellectual functions. The neuroglia

is quite frequently sclerosed either in certain portions of the brain or in its entirety.

Sclerosis and porencephalia usually predominate among the lesions encountered in the different varieties of amentia of infancy; occasionally, however, neoplasms, especially cysts, and local softening are detected postmortem in cases in which they were least suspected during life.

The cerebrospinal fluid is either increased or diminished in quantity, depending chiefly upon the size of the skull and the amount of brain structure within it.

In addition to the diverse pathologic alterations in the central nervous system, postmortem examination of mental defectives invariably discloses also several lesions in other parts of the body. The ductless glands, more particularly, the thyroid, thymus, pituitary and adrenals are often in a state of rudimentary development, hypertrophy, or degeneration. The cranial bones are either unusually thick or thin, and the diploë is diminished. The tubular bones are thick and short and often deformed. It is not uncommon to find congenital anomalies of the heart and blood vessels, and of the abdominal organs, as also malformations of the eyes, ears, palate, fingers and toes. Indeed, these anomalies are so prevalent, that they are generally accepted as special "stigmata of degeneration" (*q. v.*), and of great diagnostic importance.

Contemporary authorities are very much inclined to advance heredity to the forefront of the etiologic factors of mental degeneracy, Tredgold, for example, going so far as to claim a neuropathic ancestry in from 60 to 70 per cent of the cases of amentia. While his estimate may precisely agree with the histories of amentia housed in asylums and special hospitals for idiots, it seems to me that these percentages by far exceed those obtained in private practice. Statistics in this direction in order to be correct would have to embrace not only the personal and family history of the institutional cases (almost invariably of the worst stock and lowest class of society, and whose very life and environment are conducive to mental degeneracy), but also of the even larger number of aments who are quietly kept at home, and whose mental degeneracy is the result either of antenatal, natal or postnatal traumatism or disease, and who often succumb at an early age, not rarely long before the exact state of their mentality has at all been determined.

Those who claim the preponderance of a tainted heredity as the primary cause of menal deficiency *a priori* concur with the views of Darwin and his disciples who hold that the offspring inherit the essential characteristics of their ancestors. Now, while this doctrine unques-

tionably applies to the animal species as a whole and to the transmission of *normal* racial characteristics, I very much doubt if it conforms to the phenomena of disease, which, in contrast to normal attributes of the human species, form *abnormal*, unnatural, nay, often merely accidental accessions.

In order to obtain a clear conception of the workings of heredity, I think it best to assume two distinct phases thereof—namely, one permanent, which has become fixed during the long course of evolution; the other, temporary, accidental or transient. To the first, *permanent*, category belongs the phenomenon or hereditary transmission of normal racial characteristics. Taking the African negro, for example, we find that irrespective of the laws of variation and mutation, his offspring always maintain their racial characteristics, so long as the negro mates with members of his own clan. And even were he persistently to intermarry with descendants of the white race, there still would be little likelihood of his offspring ever entirely overcoming the attributes of their African ancestry. It certainly would require a great many generations to swamp the negro individuality, were it at all conceivable that the law of reversion would cease its vigilance and tolerate such an unnatural process of evolution. This, then, represents the permanent phase of heredity. The second, *temporary*, principle of heredity here suggested is strikingly illustrated by the transiency of certain bodily physical anomalies, as for example, supernumerary fingers and toes. These malformations are occasionally observed in several members of one family. But we usually note, that just as soon as these affected individuals intermarry with normally developed individuals, the aforementioned structural anomalies, with but very few exceptions, promptly disappear in their succeeding generations, for the very good reason that supernumerary fingers and toes are useless, abnormal and unnatural acquisitions, and hence are dropped by nature at the earliest opportunity. This phase of temporary heredity applies with equal force to anomalies of development of the central nervous system. Indeed, so anxious is nature to eliminate anomalies of development, be they physical or mental, that the great majority of degenerates are destroyed in the germinal, embryonic, fetal, or early postnatal stages of life, or if they happen to survive, are usually rendered sterile, in order to prevent the procreation of their kind.

With these considerations in view, I cannot help but hesitate to believe that heredity really plays so important a rôle in the propagation of mental deficiencies as is generally supposed, and am inclined to place much more responsibility upon acquired etiologic factors. This

reasoning is partly corroborated by the investigations of Scholomo-witch, Keller and Diem who found that the difference in the degener-acy ratio among the offspring of sane and insane ancestry is only about 10 per cent in favor of the former.

The fact frequently cited that on rare occasions (*e. g.*, the famous, or rather notorious Jukes family) we do meet hereditary mental de-generacy in several generations, does not in the least controvert the here proposed modification of Darwin's theory. In fact, in a way it even confirms it, since it can readily be shown that, as a rule, mental degenerates persistently mate with individuals of similar mental caliber (for anyone with sound mind could hardly be induced to mate with an idiot!), and therefore the continuity of intermarriage among defectives generates the phase of permanent heredity previously spoken of; in other words, a new race, as it were, with mentally deficient characteristics, is created which does for some time and would forever transmit its de-generacy to its offspring, were it not exterminated by nature in accord-ance with the law of natural selection and destruction of the unfit.

The *modus operandi* of hereditary transmission is still veiled in deep mystery. It is generally assumed that in mental degenerates the germ plasm of the male or female, or of both, is defective either in the num-ber of its component cells, in their strength, or shape, and in consequence fails to form the impetus essential to normal development of the brain. It is further postulated that under certain as yet mysterious conditions the germ cells of the opposite sexes, at the time of their fusion, are capable to influence each other, either for good or bad, in accordance with the laws of natural selection. In all probability a tainted germ plasm is deficient in more than one of its elements, since the degenerate brings into the world not only a deteriorated brain but quite frequently also several anomalies of other parts of the body, *e. g.*, abnormal heart, extremities, etc. Furthermore, there is ample reason for the belief that the anteconceptional deficiencies in the germ plasm which are produc-tive of amentia in the child may be the result not only of neuropathy in the parents, but also of other pathologic states, more especially tuber-culosis, cancer, syphilis, and the like, the toxins of which act as poison-ing and deteriorating agents upon the germ cells, the embyro and fetus, and arrest their normal development. Statistics are greatly at variance as to the exact number of the feebleminded children procreated by par-ents thus afflicted. The family histories obtained are almost always inaccurate, since but very few parents are willing to admit or are aware of the prevalence of latent tuberculosis, syphilis, etc. amongst them. Moreover, it is only with the evolution of the Wassermann and

tuberculin reactions that the statistics pertaining to the causes of feeblemindedness have at all become reliable.

All observers agree that parental alcoholism forms a most potent predisposing cause of mental degeneracy in the offspring. In 1901 the New York Academy of Medicine undertook a careful investigation of the effect of parental intemperance upon their children. The family history of 3,711 school children through three generations was traced with considerable detail, and it was found that the children of temperate parents exceeded in proficiency those of heavy drinkers by about 70 per cent, and that a large number of the encumbered children were mentally deficient to a very high degree.

The etiologic relation of consanguinity to amentia is still subject to controversy. It undoubtedly greatly depends upon the physical and mental condition of the individuals concerned. However, it has often been observed that all hereditary predispositions to disease in the parents are markedly intensified in the offspring. Deaf-mutism is particularly prone to occur as a result of union of near relatives.

The postconceptional causes of mental deficiency acting upon the embryo and fetus are as prolific as, and possibly more so than, those exerting their influence through heredity. Notwithstanding the purity and the normal activity of the parental germ plasm, it may yet fail in its destiny, if the soil in which the seed is to grow is lacking in the essential prerequisites for healthy growth and development. Let me briefly enumerate the various intrauterine morbid conditions which tend to disturb the normal progress of the embryo or fetus—in one case, e. g., acting harmfully upon the extremities or heart; in another, upon the central nerve system:

1. Disease of the uterine tissues surrounding the impregnated ovum preventing uniform contact between the maternal and embryonic structures and facile absorption of nutriment.

2. Internal or external violence acting either directly or indirectly upon the fetus.

3. Intra- or extrauterine excessive pressure hampering the commodious and equable expansion of the rapidly growing fetus.

4. High degrees of toxemia from febrile affections or poisoning from slow morbid metabolic processes, e. g., typhoid, tuberculosis, and diabetes, especially during the early period of pregnancy, may greatly affect the fetus, and finally,

5. Serious domestic trouble, grave mental anxiety and extreme fright with prolonged agitation during the early stages of pregnancy may so undermine the general health of the mother as to disturb indirectly the normal processes of growth and mental development.

In this connection it is not amiss to emphasize also that many of the dystrophies, especially of the brain, not rarely observed in prematurely born infants, are the direct or indirect result of some microscopic or gross pathologic changes either in the thyroid, parathyroids, thymus, adrenals or the hypophysis originating at an early period of intrauterine life.

There still remains another large group of mentally deficient infants who though apparently normal until birth, show definite manifestations of amentia some time thereafter. Traumatism during delivery has always been recognized as a highly potent factor in the production of idiocy and the allied mental deficiencies, the statistics relative to these cases ranging anywhere between 15 and 30 per cent. Where the cranial bones are fully developed and the maternal pelvis is free from extreme contraction or deformity, it is doubtful whether tedious labor *per se* is responsible for mental deficiency developing during early childhood. On the other hand, forcible instrumental delivery of a soft skull impacted in a narrow rickety pelvis is bound to effect some injury to the brain and leave behind a permanent mental deficiency in the child, more especially if the parietal and frontal lobes sustain the brunt of the injury. Occasionally severe asphyxia neonatorum is traceable as an immediate cause of amentia, undoubtedly owing to suggillation of and quite severe hemorrhage in the meninges and even in the brain that often accompany prolonged asphyxia. Amentia, following natal traumatism, not rarely makes its appearance several months or years after the injury has been received, and is often preceded by epileptic convulsions which are attributed to all sorts of immaterial causes. Traumatism in early infancy is an especially frequent cause of mental degeneracy in children of the slums, whose parents, either for want of means or of good sense are very apt to leave their small children to shift for themselves, so that knocks, falls and bruises form part and parcel of the miserable lot of their unfortunate babies. Apparently ''the Lord takes care of the helpless children,'' for were it otherwise the hordes of idiots would have swelled beyond calculation or imagination.

Febrile affections, more particularly meningitis, encephalitis and exanthemata form very material etiologic factors of permanent degeneration of the infantile central nervous system. Acquired hydrocephalus supervening upon grave gastroenteric intoxication, severe attacks of pertussis (by inducing cerebral hyperemia or local hemorrhage), and acquired diseases of the thyroid (*e. g.*, endemic cretinism) most probably stand next in frequency as etiologic factors of amentia. Not rarely also mental backwardness is traceable to deprivation of senses, such as vision and hearing, particularly if these unfortunate children are not

given the benefit of expert treatment and training. Several authors mention malnutrition, rachitis and adenoids as rampant causes of mental deficiency in young children. The mental dulness, however, in these cases is only temporary, promptly giving way to full vigor upon removal of underlying, mentally retarding, factors.

Diagnosis.—After reviewing the aforementioned intricate causes of amentia, we can readily appreciate the importance of obtaining a clear personal and family history of the case in question. It is especially essential to learn whether the amentia is congenital or acquired, since it furnishes the most reliable clue to the prognosis and treatment of the case. In taking the history, however, it is almost equally important to remember that histories obtained from parents are not always reliable, first, because the latter are rarely very certain of their own mental shortcomings, and more especially of those of their ancestors; secondly, they are usually loath to admit degeneracy in their immediate family; and thirdly, either for want of good judgment, or in the hope of favorably influencing the doctor's opinion, they are very apt to conceal certain mental inferiorities of their infants or to exaggerate their mental powers and thus to mislead the examiner. However, unreliable as the history may be, it always furnishes at least a few threads of information which help to direct our attention to some mute points in the diagnosis, which otherwise would escape our observation.

The taking of the family history should include questions as to insanity, idiocy, dipsomania, syphilis, tuberculosis, cancer, epilepsy and monstrosity in the immediate family, both on the father's and mother's side. The condition of the previously born children, if any, at the time of birth and thereafter. Diseases of the mother immediately before and during pregnancy. The mental state of the mother during pregnancy, especially as regards grief, fright or extreme emotion from other causes. Traumatism during pregnancy, possible means used to abort, drug habits, etc.

The past and immediate history of the patient should furnish us all details as regards asphyxia, bleeding from nose and mouth and injuries during labor. Appearance of the head and other portions of the body immediately after birth. Convulsions at this time or at any time thereafter. Mode of feeding and physical progress of the child. Diseases it suffered from, particularly as to exanthemata, pertussis and otitis. Traumatism and its immediate consequences. The period at which the infant was able to hold its head erect, to sit up, to stand and to walk, when the teeth made their appearance; and also the age of the baby when it made the first attempt to speak. It is also advisable to let the mother relate in her own way what she observed of the mental acumen of her

child, more especially in reference to its progressive or regressive character. This is important, as we intend to show later that some idiots, e. g., cretins and amaurotics, get more stupid as they grow older.

Lest we forget, let me state right now that while the parents are busy relating their ''experiences'' and responding to questions, and the patient is still in a passive mood unmolested and unaroused by the ordeal of the physical and mental examination, the physician should avail himself of the opportunity to note the attitude and behavior of both the parent and the child and ''to size up'' the general aspect of the case. Indeed, as with increased experience we gradually learn to see aright, it is often surprising how easily we can arrive at a correct diagnosis by mere superficial observation of the patient. This statement is not intended to convey the idea that such momentary examination should suffice to express a positive diagnosis. Quite the contrary ; irrespective of what impression we gain at a glance, we must never omit a very careful and minute physical examination of the child and; this completed, to apply all the mental tests presently to be outlined. But I do desire to lay special stress upon the importance of training ourselves to see a great many things at a glance. After undressing the child we proceed with the usual physical examination of children, but devote a little more attention to inspection and mensuration (see p. 173) which enables us to reveal the pathognomonic signs of amentia and more particularly the *stigmata of degeneration* which are invaluable in the differential diagnosis between congenital and acquired amentia, and should invariably receive careful consideration. Therefore let us briefly enumerate them.

Stigmata of Degeneration

1. Abnormalities in the size and shape of the head. Softness or bossing of the cranial bones. Marked gaping or premature closure of the fontanelles and sutures. Undue distention or sinking of the fontanelles.

2. Malformations of the ears. Irregularity in size. Undue prominence or flattening. Misshaped helix, antihelix, tragus, antitragus and lobule. Supernumerary auricles, auricular appendages or atresia auris.

3. Anomalies of the eyes or lids. Drooping of one or both eyelids. Epicanthus and palpebral fissures. Congenital cataract, coloboma iridis or irideremia. Micro- or anophthalmus. Strabismus, and nystagmus.

4. Malformations of the nose. Saddle-shaped, exceptionally small and broad, or unduly large and prominent. Partial or complete atresia of the posterior nares.

5. Malformations of the face. Undue prominence of the cheek bones with markedly retracted small chin. Clefts of face and lips. V-shaped or high vaulted palate. Enlarged protruding and often cracked tongue. Irregularly shaped and implanted teeth, deficiency or excess in their number. Inability to bring jaws closely together owing to irregularity of dental arches, hence constant dribbling of saliva from half open mouth.

6. Malformations of long bones. Curvatures of the bones of the upper and lower extremities. Supernumerary fingers and toes or deficiency in their number. Syndactylism or fan-shaped distribution. Disproportion in size of legs and arms. Talipes, spina bifida, and caudal formations.

7. Umbilical hernia: diastasis recti abdominis. Anomalies of the genitalia; epi- and hypospadias. Malformations of the rectum and anus.

Valuable as a tainted history and the existence of stigmata of degeneration are as diagnostic aids in amentia; they are at best only of relative value in estimating the mental state of the child in question. It is not at all unusual to meet with perfectly normal children who present a neuropathic history and several bodily malformations, and *vice versa*. Furthermore, infants of certain Mongolian races naturally possess the typical Mongolian facies and yet may be fully as intelligent as, and possibly more so than, a child of the purest white race with an unblemished history and anatomy. Hence, before declaring an infant mentally deficient, it is absolutely indispensable to put it through definite physical and mental tests, which reveal the mentality of children of certain ages and permit not only the distinction between the normal and abnormal mentality, but the degree of mental deficiency as well. The importance of such an examination becomes especially evident when we bear in mind the fact that some infants are merely slow in their mental development as a result of diseases or faulty environment, but promptly unfold their mental powers under proper care and treatment.

In order to be able to estimate the mentality of an infant correctly, we must, of course, have a perfectly clear conception of the normal intelligence at different periods of its existence. We shall, therefore, endeavor to depict the normal mentality of the infant before attempting to outline the mental tests for one who is less gifted.

Normal Intelligence

According to the latest investigation, a normal baby can hear and see immediately after birth. He feels pain when he is hurt and cries when he is uncomfortable or hungry, and exercises his extremities and the

musculature of other portions of the body,—if not immobilized by an overabundance of coverings, or fancy frocks and frills and bows and strings.

At one month he begins to locate the direction of sound and momentarily to follow a bright light.

At two months he responds to snapping of the fingers, follows bright objects more or less intently, and rejects ill-tasting food or drugs.

At three months he holds his head erect, and can turn it steadily from side to side; he smiles when accosted, shows an inclination to grasp bright objects displayed in front of him, and coos when in good humor.

At four months he begins to recognize his mother or nurse, or those who fondle him; manipulates things put in the hand, e. g., a rattle; plays with his fingers, and brings everything to his mouth.

At five months he knows his mother, nurse or father, and puts out his hands to be taken when they approach. When crying from hunger he stops promptly as the food is brought near, and opens his mouth—ready for the prey.

At six months he is interested in his surroundings; sits up in a chair with slight support; shows gratification when taken outdoors.

At seven months he recognizes familiar faces from a distance; grasps after objects placed at a short distance; begins to imitate sounds and syllables;* laughs aloud, and smiles to everybody, and cries when scolded.

At eight months he attempts to stand if held erect or to creep if placed on the floor. He is often able to repeat "mama" or "papa," to clap hands, to shake bye-bye and to perform similar little "baby tricks." He understands several words spoken to and enjoys a game of "peep bo" and the like.

---

*N. J. P. Van Baggen (of the Hague, Holland) distinguishes different periods in the development of the infant's speech. In the first period during the first year, the infant utters involuntary sounds, which must be considered as a simple muscular action of the apparatus of speech produced by an unconscious reaction of the numerous stimuli which the child receives from without. Later on the stimuli, becoming more intense, reach, through the spinal marrow, the centers of the cortex, and the child begins to feel the muscular movements and to be conscious of them. About the same time, however, the child begins to hear the sounds he produces. Henceforward he feels those sounds as well as he hears them. Both sensations now leave their traces on the cortex of the brain in those parts which are destined for the motor center of the muscles of articulation and for the center of hearing. The sensation of feeling, and that of hearing the word, occur simultaneously and therefore they become united by simultaneous association. The child now begins to imitate the sound he produces himself and soon thereafter he notices the sounds produced by others and he tries to imitate them.

The child now commences to appreciate the conformity between the sounds he hears and the sounds he utters; the imitation becomes more and more complete; and finally, syllables and simple words are pronounced. However, this pronouncing of words has not yet any meaning for him. It is only gradually that the association between the heard and pronounced word and the realization of its significance takes place. This association is brought about by the simultaneous hearing of the word and the seeing of the object which the word indicates. Whenever the child sees his doll, the word doll is repeated till at last the child unites the word doll inseparably with the object itself, and henceforward the heard word and its characteristic meaning are fixed in the child's memory. The child enters the third period when he begins to use the words which he knows by memory. When he wants his doll, he will pronounce the word even without seeing the object or hearing the word pronounced by standers-by.

At nine months he knows his name and also turns in the directions of other persons who are accosted. He easily holds and carries his bottle to the mouth; is able to bite off and masticate solid food. If properly trained, he indicates his desire to urinate and defecate.

At twelve months he stands alone, or by holding on lightly to a hand or chair, and in the same manner attempts also to walk. He knows the difference between the articles of food he is accustomed to eat. He throws a kiss or actually kisses.

At fifteen months he makes himself thoroughly understood either by signs and motions, or by baby language. He can point to the nose, eyes and ears, etc. He is interested in picture-books, colors and different toys; can turn pages and scribble with chalk or pencil. He knows the difference between a cat and a dog, and is often able to name them from life or drawings. He can play a toy piano or mouth-harp.

At eighteen months he usually runs about freely and engages in several games, such as throwing balls, marbles and the like. He can imitate all sorts of performances, such as dance, jump, hide, rock a doll, etc. He knows the difference between right and wrong, and obeys or rebels.

At two years he knows exactly what he wants in the way of food or toys, and as a rule, is able to call for them. He speaks with characteristic gestures; is able to feed himself, and to distinguish manifestations of the weather (snow or rain).

At two and a half years he can make himself, as it were, useful around the house, i. e., do little errands. He begins to ask questions and to ''show off.'' He recognizes different colors, shows constructive ability by making correct use of building blocks, etc.; carries simple tunes and memorizes more or less lengthy nursery rhymes.

At three years he uses the personal pronoun in conversation. He shows an inclination and some ability to dress himself. He can indicate the seat of pain or annoyance. If instructed he can count up to ten, at least, and spell simple words, or pick several letters of the alphabet.

The physical and mental activity of the child here depicted pertains of course to that of average normal intelligence. Some infants excel others in certain capacities, and *vice versa*. It is not at all uncommon, for example, for some babies to walk and to talk at one year of age, or conversely, barely to begin either at two years or even later, and yet be perfectly normal in every other respect. But we must set before us a standard of the average and not of the exceptional baby, and with due allowance for delay of development as a result of disease or lack of training, compare the physical and mental activity of the baby under examination with that of the assumed normal standard.

Judging from the foregoing discussion, a normal infant is supposed to acquire the power of seeing, hearing, taste and touch when he reaches the first four months of his life; attention, voluntary motion and perception during the second four months; imitation, speech and understanding in the third four months, and gradually, from month to month, to unfold and to strengthen these qualities, so that at the age of about three years he has developed into a real human being intellectually. Let us now attempt to analyze those qualities as they are manifested in mentally deficient children and to suggest workable mental tests to facilitate their early recognition.

THE ABNORMAL BABY

Vision.—As a rule idiots gaze vaguely into empty space or irregularly rotate their eyes in all directions. They rarely follow a bright object placed before them and it is almost impossible to fix their attention upon one point for more than a few moments. In testing their power of vision, however, we must assure ourselves of the absence of congenital or acquired obstruction to vision, e. g., congenital cataract, large staphylomas and the like. Sollier maintains that blindness is encountered in from 7 to 8 per cent of idiots. The importance of an early ophthalmoscopic examination of the eyes cannot too strongly be emphasized, since by this means only are we able to detect optic atrophy, symmetrical changes in the macula, and choroid tubercules, which are often decisive in the diagnosis of amentia of cerebral origin.

Hearing.—The sense of hearing is easily tested by starting some sort of a noise (ringing of a bell, clapping of the hands) while the patient is unawares. The ament who hears will ordinarily be startled by the noise, at least momentarily, even though he usually fails to turn in the direction of the noise. Some aments, e. g., amaurotics, are often violently startled by the slightest clapping of the hands. Deafness in connection with amentia is a rare congenital anomaly and almost never forms the sole cause of true mental deficiency. (See p. 730.)

Sense of Taste and Smell.—One of the very earliest signs of amentia is obtuseness or perversion of the sense of taste. Aments either chew everything put in their mouths, regardless of its disgusting taste, or conversely, spit out the most pleasant delicacies, because of their inability to detect their agreeable taste. They relish quinine as greedily as sugar, or refuse both. This perversion of taste explains why some aments are gluttons and others again barely eat enough to sustain life. The sense of smell is equally affected, but cannot be tested with any degree of exactitude until the child has attained considerable intelli-

gence. Some clinicians record lack of local or reflex response to irritating odors, such as ammonia. In these cases, however, we are most probably dealing with malformations of the nose (*e. g.*, atresia), so that the strong odor does not at all reach the olfactory nerve.

Sense of Touch, Pain and Temperature.—Almost all confirmed idiots are insensitive to pain and temperature, hence are frequently seen burnt, bruised and bitten without showing any signs of discomfort. Indeed, some of them delight in mutilating themselves. It is of daily experience to find a mentally deficient child squatting on the floor, bed or chair, rocking to and fro, diligently cracking his fingers or biting his hands, often until they bleed, and rebelling and howling if interrupted in his apparent state of enjoyment. So characteristic and im-
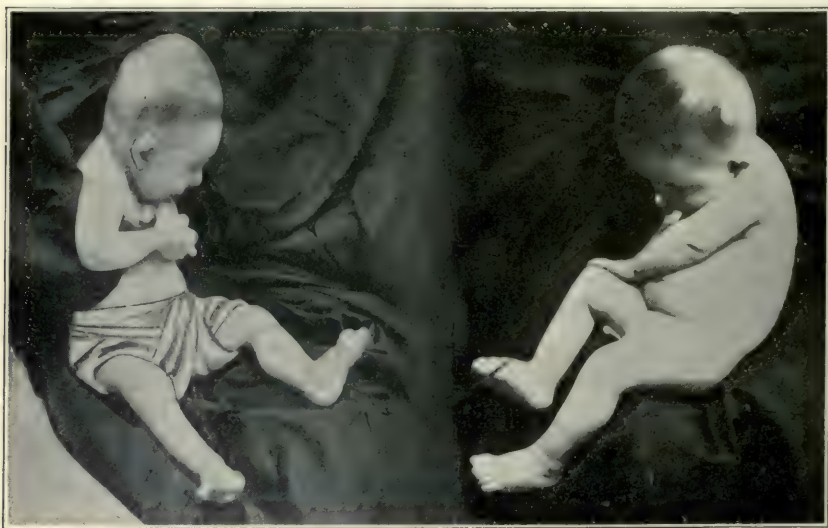
Fig. 206.—Microcephalic idiot. Status Idioticus. Fig. 207.—Amaurotic idiot.
(Peculiar attitude assumed by idiots in sitting posture.)

pressive is this peculiar attitude of the ament that a few years ago I ventured to describe it as the ''Status Idioticus'' (Figs. 206 and 207). In congenital amentia there is frequently general anesthesia, while in acquired cases the anesthesia is not rarely localized over large areas of the body, more especially in connection with paralysis. Tactile sense is not nearly as obtuse as that of temperature or pain, in fact, some aments, like the blind, show a distinct hyperacuity of tactile sensibility, being able by mere touch to recognize the individuals who take care of them.

Attention.—No other defective mental action so readily betrays the mental incapacity of an infant as his lack of power of attention. As already stated, a normal infant barely three months old, shows his

power of attention by turning in the direction of the sound of a bell, for instance, and watches the course of a bright object slowly passed before his eyes. The ament of a much more advanced age, on the other hand, is entirely unconcerned about what is happening around him. He may suddenly start when frightened by a flash of lightning and he may be aroused from his lethargic state by the approach of one who takes care of him, but he immediately falls back into his callosity just as soon as the artificial agitation has subsided. He is entirely devoid of initiative and spontaneity, and may for hours sit huddled up in one spot as long as he is not disturbed from sucking his thumbs.

Perception.—This utter incapacity of attention, of course, goes hand in hand with dulness of perception. The less attention the ament pays to the doings and actions of others, the fewer are the impressions that reach his brain, and the less capable is his cerebrum to perceive outside impulses. Moreover, his memory is so flighty that he is unable to treasure up for future use the impressions he receives. Again and again, for example, will idiots suffer pain from the effects of burns or other injuries, and yet when exposed to the same or similar harmful forces, they will not at all attempt to guard themselves against injury, for the very reason that from one time to another they forget what happened to them under such circumstances. They rarely recognize familiar faces and cannot differentiate one object from another unless specially trained in this direction.

Imitation.—In view of faulty memory, attention and perception, it is hardly to be expected that a degenerate of this sort would be capable of imitation. It is true, some of them do perform little tricks after repeated training, more especially when encouraged by mother or nurse, but their activity is extremely limited, and their performance very awkward. Unlike normal infants they do not "show off" spontaneously. Very often after learning one movement they keep on repeating the same almost indefinitely, or until they have managed to learn something else to replace it. The same lack of power of imitation hinders them from engaging in any kind of games, and, later in life, to learn to read or write, or to acquire mechanical skill to practice a trade, although, exceptionally, some aments do show considerable constructive talent and ingenuity.

Voluntary Motion.—Profound amentia is invariably associated with muscular insufficiency and incoordination. Not only are mentally deficient infants lacking in initiative to grasp objects displayed before them, but even if objects are placed in their hands, they are usually incapable of getting a firm hold of, or to manipulate, them. As a rule, they are unable to measure distance; hence, like the blind, they feel

their way in different directions, if they ever manifest a desire to locate a certain object. Amentia is frequently accompanied by paralysis of the extremities, but even in its absence aments very rarely begin to walk before two or three years of age, chiefly because they are slow to learn the special voluntary movements required in the primary act of walking. In a similar manner they rarely learn to feed themselves with a spoon; they are sure to spill its contents before bringing it to the mouth. Quite a number of aments seem to experience considerable difficulty also to manipulate the tongue, which possibly explains their frequent inability to masticate solid articles of food.

Speech.—Marked delay to walk as well as to talk is almost pathognomonic of amentia. Occasionally, a mentally deficient infant may succeed in repeating a few single short words at an early age, but he is never able to pronounce correctly several words in succession so as to form an intelligible sentence. Some aments, as they get older, keep on chattering incoherently and without measure, but they are no wiser than the others who never utter a single syllable. Hence in judging the mental capacity of the idiot, it is not the number of words he can pronounce that counts, but the way he speaks and what he says. Aments often bring out the words in staccato fashion—slow, broken or "scanning," and having, as a rule, an imperfect image of words, cannot pick the right words for the particular things they desire, and therefore fail to make themselves understood.

Intelligence.—All the aforementioned attributes of the brain collectively serve to mould the human intellect as a whole. But an infant may be able to see and to hear, smell and taste, pay attention, imitate, perceive outside impressions and, finally, to walk and to emit sounds, and yet not be endowed with normal human intelligence. Practically every domestic animal possesses these faculties. The human mind differs from that of the lower animal by its acquired faculties (not instinct) to distinguish right from wrong, to reason, judge, associate ideas, and to act spontaneously from previous experience. Now, while a properly trained infant, let us say of three years, fully appreciates that he is wrong to get soiled, reasons how best to avoid punishment, *e. g.,* by putting the blame on someone else, associates ideas, by looking for paper when you hand him a pencil; uses judgment, by not attempting to cross the street when seeing an automobile approaching, and finally, shows spontaneity and power of imagination, by making use, for example, of a box, string and cane, to take the place of a horse, whip and wagon, the mentally deficient child is utterly lacking all these mental capacities and performs certain actions only automatically by imitation after persistent training. Of

course, not all aments are alike in their mental acumen. We must always bear in mind that there are different degrees of amentia, just as there are variously gifted normal infants. But whereas the normal child through outside influences easily and readily acquires certain mental qualities as he gets older in months and years and experience, the abnormal child, owing to some faulty congenital or acquired anomalies of the brain is unfolding those mental powers at a very much later age, if ever. And it is with the object in view to determine to which period of life the mental capacity of the infant under examination— as compared with the average normal child—corresponds, that we shall presently endeavor to outline helpful mental tests for our guidance.

Mental Tests*

Mental Age, Six Months.—Move bright object in front of the child; note if he follows it. Ring bell at a distance of about 2 feet from the baby; note if he turns around.

Prick the baby's skin lightly with the point of a needle; watch for prompt facial expression of annoyance.

While the baby drinks his milk mixture, remove the bottle from his mouth and substitute a bottle containing a trace of quinine, salt or nux vomica, or warm water. Note how he takes any of the solutions. The normal baby shows the possession of the sense of taste by promptly refusing even the plain water.

Hold the baby's food at a short distance; watch the baby's facial expression of satisfaction and desire to grasp the bottle or breast. Let the mother leave the room and return from another direction; note promptness of attention.

Put the baby on the mother's lap and note his power to hold his head erect and to sit up with but slight support.

Mental Age, Twelve Months.—While unawares, call infant from a distance; note if he turns in the direction of the voice.

Put a colored object in the baby's hand, then place in front of him some article of food the baby is especially fond of; note if he drops the toy and reaches out for the food.

Let the mother encourage her baby to clap hands, shake "bye-bye" and perform similar "baby tricks"; note its power of imitation.

Mental Age, Eighteen Months.—Engage the baby in simple games, such as throwing ball and the like; note his dexterity.

---

*In infants of a year or older it is preferable to let the mother apply the mental tests, lest the child be unduly disturbed by a stranger.

Hand the baby a pencil and some article of food; note his understanding of their use.

Let the mother encourage the baby to repeat "papa," "mama" or similar words; note his power of articulation of syllables and words.

Mental Age, Two Years.—Learn whether the baby knows his own name and that of his mother, brother or any other member of the family.

Hand the baby some article of food; note his power to bite and masticate.

Put in front of the baby some constructive toy; note its power to manipulate the same, *e. g.*, to "build a house" of wooden blocks.

Ask the baby to point to his nose, mouth, eyes, etc.; note promptness of response.

Mental Age, Three Years.—Encourage baby to repeat several numbers or short nursery rhymes he was taught to recite, or to sing; note his power to memorize.

Place the child in front of a window and let him tell you what he sees on the street; note his ability to distinguish men from animals or objects.

Show him a picture-book with different animals and ask him to point to a horse, cat, bird, etc.; note the ease of response.

Display several pictures of relatives and let him pick those of parents.

Direct him to bring you different small objects from the bureau drawers or closets: note his way of going about it and the ease with which he locates them.

If already instructed, ask him to spell his name, to count, etc.; note his memorizing power.

Mental Age, Four Years.—Test his ability to feed himself with spoon or fork.

Let him reply to the following questions: Where do you live? Where do you sleep? What did you have for luncheon today, or, possibly, yesterday? How old are you? Almost all normal children of four years are able to respond promptly to these questions, or to similar ones.

Let him pick out several letters of the alphabet, especially those required to spell his name; note the ease with which he accomplishes it. He is usually able to do it, if previously entertained with toy alphabets.

Classification

In addition to these simple tests which serve to establish the diagnosis of amentia in general, we have a number of pathognomonic clin-

ical syndromes which in view of their usual occurrence with certain lesions in the brain and ductless glands, enable us to classify amentia in the following distinct clinical groups: Amentia symptomatic of microcephalus, hydrocephalus and cerebral hemorrhage and inflammation; amaurotic family idiocy; mongolism; cretinism or myxidiocy, infantilism and mental retardation (moramentia) from other causes all of which will presently receive full consideration.

Fig. 208.—Microcephalus—miniature brain.

Microcephalus

From a large number of cases under observation I have been tempted to distinguish two forms of microcephalus. One, in which the brain as a whole is very miniature, but not deficient in its component parts, thus showing arrest of development, but not a state of disease. The second variety is characterized by an absence or degeneration of several components of the brain, such as the peduncles,

pyramids or even an entire hemisphere. In these cases there may even be an hydrocephalus in conjunction with the microcephalus. In the first variety of microcephalus (Fig. 208) the skull is thick, very small and sometimes deeply furrowed. The cranial sutures are effaced and the fontanelles completely ossified. In the second variety (Fig. 209) the reverse may be the case. Indeed, in some microcephalics the skull may be moderately large and irregular in shape ("dome" or "sugar-loaf"—see Fig. 206). In microcephalics there is often also a hypoplasia of the spinal cord, more especially of the pyramidal tracts and the columns of Goll.

Fig. 209.—Microcephalus—brain degeneration.

Where the brain is intact but miniature, there is general inactivity of the cerebrospinal system. The child is entirely helpless during infancy, but occasionally gradually improves physically as he gets older. As the cranial bones are completely ossified, and the immature brain no longer has the facility to develop, the mental faculties of the child remain permanently in an infantile state. On the other hand, in the second variety of microcephalus the mental and physical condition of the child depends entirely upon the pathologic alterations of

the brain. Where the motor area is involved, we have disturbance of locomotion, convulsions, athetosis, rigidity and many other symptoms that usually accompany cerebral lesions. The mental state of the child ranges from feeblemindedness to profound idiocy. As these children get older, they are usually obstinate, vulgar and very irritable. Some of them understand simple words addressed to them and are able to imitate certain actions after prolonged training. They may learn to feed themselves, to do little errands, and possibly to help in some trade under the guidance of a master. The majority of them, however, are entirely devoid of understanding and take no interest in their surroundings; and especially while under three or four years of age, they may for hours sit or lie in one position and indulge in irregular movements, without by attitude or facial expression indicating any desire for a change or even betraying any discomfort during or after defecation or urination. Owing to their extreme restlessness and awkwardness of locomotion, their grotesque movements, in hopping from place to place, often resemble those of rabbits, goats or monkeys, and in times bygone they were exhibited by showmen as curious descendants of a lost degenerated tribe. Some of them are witty and alert and show distinct powers of mimicry, but they never attain a sufficiently high degree of intelligence to earn a livelihood independently.

The diagnosis of microcephalus is based principally upon the size and shape of the head. In the first variety of microcephalus, where owing to early arrest of development of the brain the cranial bones ossify before or immediately after birth, the circumference of the skull always remains from 3 to 6 inches below that of the average normal child. To a slighter extent (2 to 3 inches) this is true also of the second variety of microcephalus. The hair is often so coarse and wiry that as Tredgold puts it, the teeth of the clippers often break whilst the hair is being cut. A microcephalic idiot may sometimes be mistaken for a Mongolian. In *mongolism*, however, the head is not quite as small or malformed, the hair not as coarse, and the muscular flaccidity or rigidity not quite as pronounced, while in microcephalus protrusion and cracking of the tongue is exceptional. In early infancy the mentality of the Mongolian is on a higher plane than that of the microcephalic idiot. The flaccid type of microcephalus may occasionally resemble *amaurotic family idiocy*. In the latter condition, however, there is usually a history of gradual degeneration after birth; the fontanelles are usually open, the size and shape of the head fairly normal, and an ophthalmoscopic examination reveals pathognomonic changes in the retina (*q. v.*). The aforementioned symptoms of microcephalus are also

ample to differentiate this form of amentia from that associated with traumatic cerebral palsy. Besides, in *paralytic amentia* of natal origin, congenital stigmata of degeneration (*q. v.*) which almost invariably prevail in microcephalus, and the "idiotic grunt"—the gutteral noise which the microcephalic usually exhibits particularly when he is enjoying a square meal—are usually absent. Finally, it is well to bear in mind that "sugar-loaf" head (oxycephaly) is occasionally met with in perfectly normal children.

Hydrocephalus*

The pathologic anatomy in hydrocephalic amentia varies greatly with the quantity of cerebrospinal fluid in the cranial cavity, the

Fig. 210.—Hydrocephalic idiot.

period of its appearance and the length of time it has continued to exert pressure upon the vital structures of the brain. Thus do we find that in cases of postnatal hydrocephalus where the pressure happens to be slight and temporary, the pathologic alterations in the brain are often insignificant, whereas in marked congenital hydrocephalus

*For "Acquired, Acute and Chronic Hydrocephalus," see p. 596.

postmortem examination usually reveals considerable atrophy of several parts of the brain. The brain markings are generally effaced, the ventricles distended and their contiguous structures compressed and degenerated. The meninges are thin and bulging, the cranial bones greatly atrophied, and the fontanelles and sutures widely gaping with Wormian bodies freely distributed in the intervening spaces. Not rarely hydrocephalus is associated with spina bifida—undoubtedly Nature's attempt to relieve the excessive intracranial pressure (Fig. 210).

The most striking physical sign of hydrocephalic amentia is the extraordinary size and shape of the head. The head is usually asymmetrical, twisted in appearance (plagiocephalic), but may be rounded, egg-shaped (brachycephalic), long and narrow (dolichocephalic), or keel-shaped (scaphocephalic). The circumference of the head ranges between 22 and 30 inches or more. The scalp is very thin and barely covered by fine hair and traversed by conspicuous veins. The cranial bones are soft and often yield to light pressure with the finger, imparting the sensation of parchment. In severe cases the orbital plates are pushed downwards while the eyeballs protrude forward, so that the lids are more or less retracted, leaving a ring of the sclerotic exposed. This anomaly gives rise to the peculiar staring expression of the eyes which is characteristic of the hydrocephalic idiot, and is especially pronounced when accompanied by strabismus and nystagmus.

The mental symptoms are not invariably correlated to the size of the head, some infants with huge heads occasionally possessing more intelligence than those with proportionately smaller heads. And if, perchance, the hydrocephalus is arrested before permanent damage to the brain has been wrought, the hydrocephalic may yet grow up with a fair degree of mental capacity. Ordinarily hydrocephalic aments are quiet, gentle, timid, sorrowful and affectionate, and but little impressionable or curious. Owing to impaired function of the extremities by paraplegia and spasmodic contractures of the arms, they are rarely able to walk about and to help themselves, and when, as is often the case, vision (optic atrophy) and hearing are affected, they usually remain infantile for life—which latter, fortunately is very rarely of long duration. Occasionally, hydrocephalus is associated with adipositas (Fig. 171), which most probably occurs in consequence of interference with the functional activity of the hypophysis cerebri.

Chronic hydrocephalus may be confounded with rachitis, syphilis and macrocephalus in connection with hypertrophy of the brain. In *rachitis* the extremities are weak but neither paralyzed nor rigid, while mental deficiency, if present, is but slight; in hydrocephalic amentia

the reverse is the case. The rickety head never attains the size of that
of the hydrocephalic, and the cranial bones rapidly assume their nor-
mal consistency upon removal of the cause of rickets, *i. e.*, on attention
to hygiene and proper nutrition and administration of lime and phos-
phorus. In rachitis we usually find that in the first few months of its
existence the child's physical and mental condition was normal,
whereas in hydrocephalus there is a history of the presence of all of
the aforementioned symptoms from birth on, or their sudden develop-
ment in connection with some serious acute affection, especially tuber-
culous or cerebrospinal meningitis (*q. v.*). Moreover in these cases
the child rarely escapes severe involvement of the eyes and ears. In
syphilis the head is not rarely greatly enlarged, but instead of being
unusually soft it is often hard and bossed. Of course, where the
syphilitic is also suffering from hydrocephalus, which is not at all
uncommon (Fig. 137), the differential diagnosis between these two
forms of amentia can be made only by means of Wassermann reac-
tion, which should at any rate be employed from a therapeutic point
of view. The following suggestions will prove helpful to differentiate
hydrocephalus from *macrocephalus* associated with hypertrophy of
the brain.

Hypertrophy of the Brain	Hydrocephalus
The cranial bones are usually normal in consistency: the enlargement develops slowly	Usually the reverse
Marked pulsation at the anterior fontan-elle	Slight, if any
Sutures slightly disconnected	Widely gaping
Ordinarily normal mentality or only slight deficiency	Idiocy as a rule
Slight intracranial pressure—if the fon-tanelles are open	Very marked

Paralytic Amentia

(VASCULAR, INFLAMMATORY, TOXIC, MENINGITIC, OR EPILEPTIC AMENTIA)

Under this heading are generally grouped the numerous cases of
mental deficiency which are due to more or less extensive lesions in the
brain occurring either before and during birth of the child or in the
course of his first few years of life. The cerebral lesions may be the
result of hemorrhage or inflammation, or both, accompanying prenatal,
natal or postnatal cranial traumatism, asphyxia neonatorum, meningi-
tis, encephalitis, influenza, measles, scarlet or typhoid fever, pertussis
and similar microbic affections, and neoplasms. In the great majority

of these cases the cranial bones are reduced in thickness, the meninges are adherent, and some of the convolutions are compressed, atrophied and indurated. Some portions of the brain are in a state of softening, others are found to have undergone cystic degeneration, cicatricial contraction and sclerosis. The lesions are productive of variable clinical pictures in different individuals. They may lead to paralysis in one child, convulsions in another, and to amentia in the third, or to all these manifestations in one and the same child. Moreover, these phenomena are not invariably correlated to the extent of the lesions. And one is occasionally surprised to find diffuse lobular sclerosis of the brain with extensive blood cysts and porencephaly in a child who during life was apparently endowed with fairly normal mental faculties, and, conversely, only minute cerebral lesions with total idiocy. As a rule, however, in infants any seemingly trivial intracranial accident is followed by mental deficiency, hemiplegia or diplegia, and this is especially the case with lesions in the frontal, prefrontal and parietal lobes.

Of 55 cases of hemiplegia examined by Sachs and Peterson, the mental impairment was feeblemindedness in 16 children, imbecility in 31, idiocy in 7 and epileptic insanity in 1 case. In diplegia the percentage of mental deficiency is always very high, between 60 and 75, whereas in cerebral paraplegia, which condition is usually associated with less extensive lesions, the mental deficiency is rarely very pronounced. Epilepsy, especially of the Jacksonian type, is quite a common sequel of cerebral hemorrhage or inflammation and ultimately ends up with progressive amentia.

In order to obtain a clearer conception of the symptom complexes that usually follow the aforementioned pathologic alterations in the brain, it is advantageous to classify these cases in three large groups, in accordance with the time of their development, either before, during or after birth. The *first group* usually reaches this world in a more or less abnormal physical condition. These children are often prematurely born, emaciated and disfigured, and of very low vitality. The head is either small and asymmetrical or normal in size, but soft and flattened on one side. The extremities are either rigid or slightly movable, or there may be mono-, para-, or diplegia. (See Fig. 211.) As these symptoms are the result either of arrested development of the brain or cord, or of both, or hemorrhagic or inflammatory processes therein, it is not at all uncommon to find several cranial nerves implicated. Under these conditions, of course, the diagnosis of congenital paralytic amentia is self-evident. The *second group* is generally described as Little's disease, or diplegia or paraplegia spastica infantilis. (See

p. 615.)   In the great majority of these cases there is a history of natal traumatism, asphyxia, convulsions immediately after birth and other signs of acute cerebral involvement. (See Fig. 213.) These cases, in addition to the characteristic physical syndrome, frequently present mental deterioration, ranging from simple feeble-mindedness to total idiocy, and are often accompanied by stammering, nystagmus, strabismus, athetosis and epileptic convulsions. The *third group* of cases gives a history of apparently normal physical and mental

Fig. 211.—Paralytic idiot of antenatal origin.

development at birth, and of an acute or insidious onset of some febrile or wasting disease, or of traumatism some time after birth, which was later followed by amentia with or without paralysis or epilepsy, and often by degeneration of the cranial nerves. The mental impairment is usually progressive in character, and in older children may not be fully recognized until several months or years after the accident or termination of the primary affection. To this group of cases, encephalitis and meningitis and their prolific sequelæ, more especially partial or

total deaf-mutism and blindness, contribute the greatest number of victims, although traumatism with its great tendency towards epilepsy is exceedingly conspicuous in the histories of postnatal amentia recorded. According to Fletcher Beach, other infectious diseases, such as typhoid, scarlet fever and measles do not form very rampant causes of this variety of amentia, for after examining the history of 2,000 cases of idiocy, imbecility and feeble-mindedness he found only 37 (or 1.85 per cent) which could be traced to an attack of one of those affections.

In connection with paralytic amentia it is opportune to call attention to a form of mental backwardness which is occasionally encountered as a result of hereditary syphilis. As has already been stated the amentia may appear in consequence of hydrocephalus or in connection with

Fig. 212.—Paralytic amentia in consequence of cerebral hemorrhage during instrumental delivery. The baby died at the age of two and one-half years from miliary tuberculosis.

mono-, hemi-, or diplegia consecutive to syphilitic meningitis or encephalitis. More rarely the foundation to the amentia is established during intrauterine life in the form of gummatous infiltration and sclerosis of the brain. In this event the child is born with all the symptoms corresponding with the primary lesion in the brain, e. g., paralysis, defective vision or hearing, or involvement of other cranial nerves. Except for the history of the case and the positive Wassermann reaction there is practically no way of distinguishing syphilitic from nonsyphilitic paralytic amentia. However, irregular enlargement of the head, particularly Parrot's nodes, should serve to arouse our suspicion.

Amaurotic Family Idiocy*

This form of amentia is based upon specific pathologic alterations in the brain. It is characterized by some degeneration in the cerebral white fibers throughout the course of the pyramidal tracts, in the inner capsule, crusta, pons and medulla, and also of the pyramidal tracts in the lateral as well as the anterior columns of the cord. Furthermore, the same changes are found also in the gray matter of the central nervous system— in the cortex of the brain, in the cranial nerve nuclei, and in the gray matter of the spinal cord down to the lowest lumbar and sacral segments. Wm. A. Holden has further established the fact that the changes in the retina are identical with those in the brain and cord, and were due to a degeneration of the retinal ganglion cells. Hirsch, after a very exhaustive histologic examination of several cases under his care, concluded that not only are the cells of the cortex of the brain affected but the ganglion cells of the entire nervous system, the main features being a condition of chromatolysis and other degenerative processes of protoplasm, combined with considerable swelling of the cell body and displacement of the nucleus towards the periphery of the cell. The neuroglia and the blood vessels are found to be perfectly normal.

Like the pathology, the physical and mental characteristics are entirely pathognomonic. The apparently normally born and developing infant begins to fail in strength as it reaches the age of six or eight months. Although not losing in weight, nay, sometimes even gaining, it is noticed that the baby is unable to hold up his head, to sit erect, firmly to grasp objects placed in his hands, and even forcefully to suck on the nipple of breast or bottle. Simultaneously with the muscular atony the baby begins to lose interest in his surroundings, fails to smile when accosted and to follow bright objects to which his attention is being directed—all indicating mental deterioration. When the backgrounds of the eyes are examined a very peculiar retinal image is obtained. Namely, the maculæ are cherry red in color and surrounded by large grayish white patches. The optic nerves are atrophied in the great majority of cases and there is often also strabismus and nystagmus. These eye symptoms gradually lead to total blindness, and the muscular atony rapidly borders on paralysis. Hearing at first is hyperacute, but in the later stages of the affection becomes obtuse. At this time also there is often inordinate "explosive laughter," difficult deglutition and a marked tendency to recurrent convulsions. In one case I noted pronounced hirsuties over the greater portion of the body. Thus deprived of sight and partially of the sense of hearing, limp and lan-

---

*Warren Tay described this affection in 1881 as a purely local inner eye disease, while B. Sachs, in 1887, recognized and described it as a distinct brain affection.

guid as a result of the ever increasing atony of its musculature, the helpless creature gradually loses all its other senses and, fortunately, also its life. This usually occurs before the child attains two years of age. More recently, Vogt has described a ''juvenile'' form of amaurotic family idiocy which begins to manifest itself at a later age and runs a more protracted course. Its identity with the ''infantile'' form of the disease, however, is not generally conceded.

Fig. 213.—Amaurotic family idiocy in baby 14 months old. Note inability to hold up its head. (See also Fig. 207.)

Fig. 214.—Macular change (cherry-red discoloration) in amaurotic family idiocy. (After Tay.)

As its term indicates, this form of amentia affects several members of the same family or those who are closely related, and shows a very striking predilection for offspring of the Hebrew race, more especially of immigrants from Russia and Poland. This peculiar family predisposition seems to confirm the view held by Sachs and others that amaurotic

family idiocy is due to a congenital arrest of development, although the "juvenile" form seems to point to a toxemic nerve degeneration of postnatal origin.

In the early stages of the disease amaurotic idiocy may readily be mistaken for *rachitis,* but in this affection the pathognomonic amaurotic eye symptoms are absent and the mental deficiency, if there be any, is very slight. Furthermore, rachitis usually sets in more frequently in infants over ten months of age and the muscular atony of the trunk and spinal muscles is never so pronounced as to produce dropping of the head backwards. More difficulty may be experienced in differentiating amaurotic family idiocy from *cerebral neoplasms,* be they syphilitic, tuberculous or malignant. I recall a case of gliosarcoma of the pons affecting a one-year-old infant, that was under observation of several pediatrists and ophthalmologists of note and was diagnosed as incipient amaurotic family idiocy, none of them even suspecting the presence of a cerebral tumor. While in the latter affection optic atrophy is a common symptom, there is never a cherry red discoloration of the maculæ. Furthermore, in tumor the muscular atony, paralysis and convulsions are most apt to be unilateral in the beginning, and gradually bilateral, while in the amaurotic all the symptoms are bilateral right from the start. In early infancy *Mongolian* and amaurotic idiocy have two cardinal symptoms in common—namely, protrusion of the tongue and general muscular atony, which may lead to errors in the diagnosis. In such cases an ophthalmoscopic examination is decisive. It will also be found that in amaurotic amentia the tongue protrudes but slightly and inconstantly, and is otherwise normal in appearance, the reverse being the case in mongolism. Furthermore, in the latter condition the hair is wiry and the hands are usually spade-like, and the mentality deficient from birth on. As has already been stated, in all cases of doubt an ophthalmoscopic examination should invariably be resorted to before arriving at a positive conclusion.

Mongolism

Except for the proportionately undue smallness of the pons, medulla, and cerebellum in relation to the cerebrum as compared with those of normal babies, the central nervous system of the Mongolian idiot shows no characteristic lesions. As in other forms of amentia the brain is immature and its cells are imperfectly developed. With growth of the body as a whole the brain too attains a higher state of perfection, but is never capable of unfolding the faculties of normal intellect. Mongolism is frequently associated with anomalies of the thyroid gland (hence were formerly often described as "cretinoids"),

and of the heart, and not rarely with general tuberculosis. There seems to be an etiologic relationship between mongolism and syphilitic heredity. Sutherland, for example, has found a history of syphilis in 11 out of 25 cases of mongolism under his observation.

This form of amentia was first described by J. L. Down in 1866, calling particular attention to the facial resemblance of the members of this group of idiocy to those of the Mongolian, Asiatic races, such as the Chinese, Calmucks and Malays. The typical Mongolian idiot has a small egg-shaped (brachycephalic) head, covered by smooth, or dry frizzly hair; small aquiline nose, which is bound laterally towards the eyes by distinct vertical or semi-lunar folds of skin which cover the inner angle of the eye (epicanthus); triangular nostrils: almond-shaped, slanting, often prominent eyes with speckled irides and eczematous eyelids, not rarely also ectropion; flat, usually flushed expressionless face, with high cheek bones; distorted ears; high narrow palate; cracked more or less protruding tongue with markedly enlarged papillæ (later the so-called "scrotal tongue"); and irregularly set, discolored teeth. The hands are flabby, clumsy, spade-like, and the thumbs are stubby (due to atrophy of the phalanges). In addition to these characteristics the Mongolian idiot generally presents marked laxity of the articulations, so that the tips of the fingers may be hyperextended almost to touch the dorsi of the hands, and the feet may be brought up to the neck and ears while he is in a sitting posture. The little fingers are usually very thin and curved inward; the genitals ill-developed; the skin is dry, rough and hairy, and owing to circulatory disturbances the Mongolian idiot often suffers from chilblains and cracked lips which are kept raw by the dribbling saliva. He is seldom free from hypertrophied adenoids and their sequelæ, i. e., nasopharyngitis, bronchitis or even recurrent pneumonia. The latter may possibly be also due to the frequently accompanying rachitis, more especially chicken breast, large abdomen and spinal curvature.

Notwithstanding all their troubles, Mongolian idiots are of a happy disposition, placid and affectionate, and fond of music. For this reason parents often fail to recognize the abnormal state of their children, even though they note their general bodily weakness, more particularly their inability to sit erect, to stand and walk. At about two years of age these aments usually become more active, vivacious (always "on the go"), mischievous, full of grimaces and facial contortions—often misleading the parents to believe that they had outgrown their tardy development, and even to assume that their children were, exceptionally bright. However, as time goes on, it is generally found that their mentality is practically at a standstill, that

they can rarely understand when spoken to and much less are able to speak. It is not uncommon to meet with Mongolian idiots, two or three years old, barely able to repeat single syllables, to feed themselves even with the fingers (tendency to gobble down the food), or to respond to Nature's calls. As they get older they learn to walk and to make themselves understood, and after suitable training, to make themselves useful and to perform little acts for their personal comfort, but they always remain in a primitive mental as well as physical state of development; unreasonable, helpless, awkward and uncleanly, often acquiring vicious habits (*e. g.*, masturbation) which help to undermine their frail constitutions.

With this clinical picture in view, there ought to be no difficulty to distinguish typical mongolism from similar forms of amentia. Atypical cases, however, may be mistaken for cretinism, microcephalus and rachitis.

Fig. 215.—Mongolian idiot of 23 months, Calmuck type.

In *microcephalus* the idiocy is more pronounced, the head either very small or asymmetrical, and the ability to make free use of the extremities in grasping, standing and walking appears at a very much later age than in the Mongolian idiot.

Mongolism differs from cretinism in the following particulars:

Mongolism	Cretinism
Skull brachycephalic	Quite normal
Hair straight, or wiry and abundant	Fine and sparse
Skin thin, hairy and mottled	Swollen "padded"
Face flushed, vivacious	Pale and apathetic
Eyes almond-shaped; epicanthus	Palpebral fissures horizontal
Tongue narrow, cracked	Broad, swollen, pale
Little finger curved inward	Stumpy
Thyroid treatment of little benefit, if at all	Very beneficial

We can readily distinguish *rachitis* from mongolism by the fact that in this affection the head is more or less square, soft and covered by fine hair more especially along the occiput. The eyes are normal, the face is pale, the tongue is neither protruding nor cracked, and the fingers are normal in shape. Rachitis may delay the cerebral functions for a few months, but the powers of speech, perception and voluntary motion are intact, and the mentality of the rachitic child rapidly improves with the amelioration of its physical condition.

Finally, let me emphasize that a diagnosis of mongolism should not be based upon the infant's physiognomy alone, for occasionally we may be confronted by a baby of Mongolian ancestry who may be otherwise perfectly normal in body and mind.

Cretinism, Myxidiocy

Thyroid insufficiency though primarily not a brain affection sooner or later gives rise to degeneration of the central nervous system, more especially of the cortical cells. In congenital cases the cerebrum is usually considerably smaller than in normal children, and its convolutions are simplified; the cerebellum is asymmetrical and its laminæ are reduced in number. It is not rarely associated with hyperplasia of the pineal gland, the hypophysis and thymus gland, showing Nature's attempt to compensate the thyroid insufficiency by hyperactivity of similar structures. Pathologically, we distinguish two forms of thyroid insufficiency which lead to amentia. 1. Athyreosis or absence of the thyroid gland which is generally a congenital anomaly, but may exceptionally occur as a result of traumatism or accidental extirpation (cachexia thyreopriva). In congenital athyreosis the gland is frequently found replaced by cysts or other neoplasms. Occasionally degenerated (or healthy!) thyroid tissue is implanted in the base of the tongue. 2. Hypothyreosis or deficiency of thyroid gland which may be of antenatal origin (*e. g.*, congenital goiter) or develop later as a result of disease or traumatism. To this group belongs also the endemic form of goitrous degeneration of the thyroid which prevails especially in certain sections of Switzerland, Germany, Asia, England, Russia, Hungary and America, in shut-up valleys of mountainous districts, and is supposed to be due to some toxic substances in the unboiled drinking water.* That goiter is not uncommon

---

*Note.—This view has recently been disputed and a number of clinicians look upon endemic cretinism as an infectious disease. In this connection the report of A. Kutschera (Wien. klin. Wchnschr., No. 45, 1910) is of considerable interest. He relates that he found two dogs to develop cretinism who shared the bed of their mistress, a semicretin. One dog was completely idiotic, could not bark and reacted to nothing. It had dry, brittle, dirty hair, and milk teeth together with permanent teeth. After removing these two animals the author put in the cretin's bed a healthy four-months-old pup of healthy parents. After three months this pup

in very young infants can be gathered from the statistics collected by
Demme, who among 643 cases found 53 to be of prenatal origin, 37
which developed in infants under one month of age, 59 between two
and twelve months, and 35 between thirteen and forty-eight months.

Postmortem examination discloses in cretinism marked alterations in
the osseous system. The cranial bones are thickened, the diploë is di-
minished, and, according to Virchow, the sphenobasilic suture prema-
turely closed. The long bones are thick and short and often markedly
deformed. As in other forms of profound amentia there is in cretin-

Fig. 216.—Cretin from birth; total idiot. Note ''trident hand.''

ism retarded development of the centers of ossification of the carpals
and of the epiphyses of the metacarpals and phalanges. Section of the
tubular bones usually shows an invasion of fibrous tissue from the
periosteum in between the epiphyses and shaft, thus hindering the
growth of the bones in length. Around the base of the epiphysis there
is sometimes a sheath-like prolongation which may even be ossified and

developed a large head, and ten months later it became a full fledged cretin while the rest of
the litter of the same parents who were not exposed to cretinic infection remained perfectly
normal. A second animal of a large race, which could not conveniently occupy the same bed
with the cretin, also developed normally. The author, therefore, believes that cretinism is
transmissible by direct, close contact.

form a distinct cup around the epiphysis. But in contrast to what is observed in rachitis, there is no proliferation of cartilage cells near the line of ossification. The same overlapping or cupping of the epiphyseal cartilages is noted also in the ribs and innominate bones and in the scapulæ.

The physical and mental manifestations of cretinism vary greatly with the degree of thyroid insufficiency. Moreover, they set in at a later period in breast fed than in artificially fed infants, owing to the fact that during the first few weeks of life breast fed infants receive an ample supply of thyroid gland substance through the mother's milk to counteract their thyroid insufficiency. In acquired athyreoism the characteristic symptoms of cretinism usually appear gradually, but once the clinical syndrome is completed, it is practically alike in the prenatal as well as in the postnatal cases. The head of the cretin is either normal in size or slightly enlarged, flat and plump and set upon a thick, short neck. The fontanelles usually remain open, the forehead

Fig. 217.—Normal at one year.

Fig. 218.—Same case as Fig. 217 pronounced cretin at eight years.

is low, and the root of the nose is broad and sunken. The face is weak and senile. The eyelids and lips are edematous and the tongue is large and "swollen" and hence, ever protrudes from the half-closed mouth. The teeth are slow in coming and rapid in decaying. The abdomen is greatly distended, often marked by a large umbilical hernia. The extremities are more or less deformed and the articulations thickened. The hands and feet are short and flabby. Cretins learn to walk late, and their gait is awkward and draggy. The skin is dry, waxy and doughy in consistency, and the hair is sparse and brittle. The body

temperature is generally subnormal, and owing also to the ever present anemia, cretins are very sensitive to cold, notwithstanding their corpulent appearance. "Fatty tumors" are usually found in the supraclavicular and axillary spaces.

The intelligence of the infantile cretin, as has already been stated, varies with the functionating capacity of the thyroid gland. In congenital athyreosis there is total idiocy (myxidiocy). Some cretins, the so-called semicretins, possess a fair measure of intelligence. They appreciate their surroundings, and are able to acquire a meager vocabulary which may be ample to make their urgent wants understood, or

Fig. 219.—Same case as Fig. 218 four weeks after treatment with thyroid.

even to reply to simple questions. On the other hand, where the thyroid insufficiency is marked, they never reach even this low state of mental development, and, on the contrary, get more stupid as they grow older. In the great majority of cretins, the special senses are implicated. Taste and smell are obtuse; hearing is defective and vision dull. The voice of the cretin is ordinarily husky. Like the hydrocephalic aments they are timid, gentle and unassuming, and if left untreated, they retain their childish behavior for life.

One of the most characteristic features of cretinism is its marvelous improvement under thyroid feeding. After exhibiting thyroid gland extract in one form or another for but a short time, the cretin is often trans-

formed from an uncouth, apathetic and clumsy little creature into a lusty, gracile and growing human being. Thus the blurred facial features gain youthful expression; the lusterless, withered hair takes on new life; the stunted stature shoots up to almost normal proportions, and the brutal stupidity gradually gives way to human intelligence. However, this marvelous transformation lasts only as long as the thyroid medication is permitted to exert its wonderful influence. With dis-

Fig. 220.—Same case as Fig. 218 ten weeks after treatment with thyroid.

continuance of the treatment the cretin slowly but surely sinks back into his everlasting idiotic condition.

Total athyreosis in the early stages and partial cretinism at any period of early childhood may be confounded with severe forms of rachitis and *mongolism*. The differentiation of the latter form of amentia from cretinism has already been discussed in connection with the former affection. (See p. 720.) In distinguishing cretinism from *rachitis* it is well to bear in mind that the latter may complicate the former disease. But in rickets the deficiency of intellect is slight and not progressive; the tongue is neither large nor protruding; the skin is soft and thin and not rough and edematous; the hair is normal and bald

only in spots, especially over the occiput, whereas in cretinism the hair is brittle all over the scalp, and, finally, the rachitic baby learns to talk early and its voice is perfectly normal even though it may be weak. Rachitis complicated by *congenital macroglossia* and adenoids may on very rare occasions lead to errors in the diagnosis, but careful inquiry into the history of the case and the exhibition of thyroid extract will soon clear up all doubts. Furthermore, it will generally be found that in congenital macroglossia the tongue gets gradually relatively smaller as the child's mouth grows larger, which is not the case in cretinism. Besides, there is always the marked difference in the physical and mental development of these children.

Infantilism

Under this heading are generally grouped several types of abnormal infants who never attain the physical and mental development of adults and who retain several characteristics of infants and young children throughout life. In physiognomy and stature infantilism is closely allied to cretinism, and in many instances directly dependent upon thyroid insufficiency. Similar clinical syndromes have more recently been observed in connection with deficient functions of the thymus, adrenals, pancreas, and pituitary gland, and some observers claim that similar arrests of development occur as the result of systemic poisoning by the syphilitic germ and other microorganisms. In accordance, therefore, with the aforementioned etiologic factors, infantilism may be classified in the following types:

> Thyroid infantilism,
> Thymus infantilism,
> Pituitary infantilism,
> Heredosyphilitic infantilism,[†]
> Dystrophic infantilism,
> Cardiac infantilism,
> Intestinal infantilism,
> Malarial infantilism,
> Pellagra infantilism.

Two special types of thyroid infantilism are generally encountered—namely, typus Brissaud, which is characterized by fullness of the face, plumpness of the body and clumsy extremities (Fig. 221), and typus Lorain whose stature is gracile and whose facial features are pleasant and comely (Fig. 222).

The subject in question is as yet awaiting considerable elucidation. With the advance of our knowledge of the normal and abnormal actions of the ductless glands, we shall undoubtedly be able to clas-

---

[†] I recently saw a case of this type weighing only 29 pounds at nine years.

sify infantilism in two large groups, thus: Genuine infantilism, embracing all cases in which mental deficiency predominates, and a second form of infantilism, which is being described as microsomia, nanosomia, ateliosis, asthenia, achondroplasia and Herter's infantilism, in all of which physical arrest of development predominates. I may state, by the way, that contrary to what is frequently recorded in

Fig. 221.—Infantilism, Brissaud type, six years old; measures 32 inches in length.

Fig. 222.—Infantilism, typus Lorain, four and one-half years old; measures 32 inches in height and weighs 28 pounds, acts like a two-year-old infant. Note absence of left thumb and rudimentary development of right thumb.

medical literature, so-called Herter's infantilism is not associated with actual mental deficiency. To quote this author: "The intelligence of these patients was in every instance good, although the necessity of living very carefully and obeying the directions of the physician and nurse has tended to make these children somewhat introspective as regards their own ailments and to form the basis of

what might with increasing consciousness develop in after life into a hypochrondriacal condition.''

Congenital infantilism like congenital idiocy in general is often associated with physical stigmata of degeneration. Atrophy of the genitalia is particularly common where the ductless glands are involved. (See Fröhlich's Syndrome, p. 570.) As in the idiot, we often find retarded development of the carpi also in infantilism. (See Fig. 223.) The mentality of these children is very variable and depends entirely upon the period of life at which their mental development
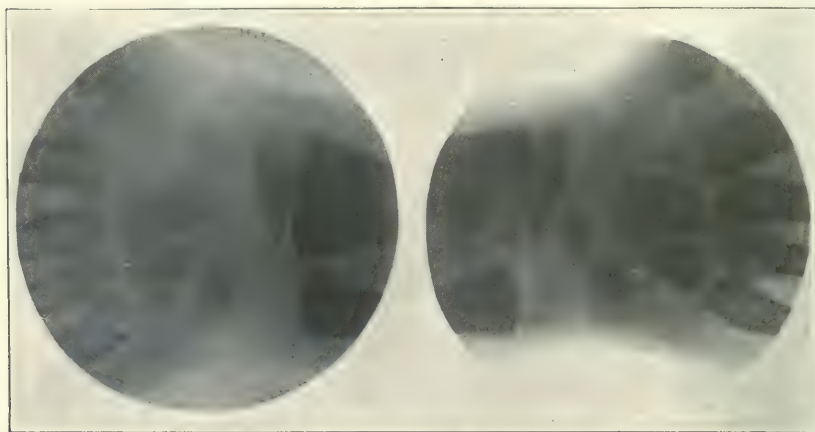
Fig. 223.—Left, wrist of ament 10 years old; right, wrist of normal child six years old. Note greater number of carpi in the latter.

has been arrested. As a rule, they are never totally idiotic, and the majority of them are able to help themselves, to walk about and to play, and to understand a simple conversation. Speech is usually delayed, but with advancing age and proper training, they ordinarily learn to speak, as well as to count, to write, and to earn a modest livelihood.

Moramentia*

(RETARDED MENTALITY)

Delayed mental development is quite frequently the result of the following causes: (1) Deprivation of special senses, e. g., sight and hearing; (2) chronic affections, such as heart disease, and other severe nutritional disturbances; (3) faulty environment and education, or isolation.

---

*For this group of cases mora-mentia would be a very appropriate term; mora (æ) signifying delay, impediment, hindrance, and mentia being used to designate mentality.

Sense deprivation as a cause of retarded mentality need not be complete. Mere errors of refraction, for example, by leaving the child ignorant of numerous objects outside its field of vision, may be entirely sufficient to delay the unfolding of its mental faculties. Similarly do we find that an infant afflicted with adenoids, which interfere with acute hearing and render it listless and inattentive, at least temporarily fails

Fig. 224.—Moramentia in a two-year-old boy, as a result of marked adenoids with its consequences, especially difficult hearing.

to receive the outside impulses to the brain and hence remains mentally backward. These children, however, are not suffering from amentia in the true sense of the word. On the contrary, experience teaches that just as soon as the retarding elements are removed, e. g., removal of the adenoids and correction of the visual defects, the supposedly mentally deficient children rapidly reach a normal state of mental development.

Defective vision, particularly if congenital in nature (*e. g.*, congenital cataract) or acquired soon after birth, forms a greater impediment to normal mental development than a similar defect in the sense of hearing. It is quite common to meet with very intelligent deaf-mutes who by means of lip-reading or dactylology even in early childhood are able to make themselves understood and fully to express their wants. Very recently two deaf-mutes, brother and sister, came under my observation, who for intelligence could pass muster as any normal children of the same ages. Their parents were first cousins, and their father was fifteen years older than the mother. The little girl was eight years and the brother two years old. They had another brother, who was able to hear and to speak, but died at the age of five years during an attack of pneumonia. It was a most pathetic sight to watch the two children by means of dactylology and lip-language to converse among themselves or with their mother, and it was astonishing how much information the mother was able to convey to the little baby. The older child was full of life and possessed of powers of observation and imagination rarely to be met with in perfectly normal children of her age. While examining her she was intently interested in everything I was doing, and as I was testing her hearing—hoping possibly to detect a trace of it intact—she concentrated her whole mind upon the test, and off and on gleefully announced to her mother that she was capable of hearing—poor child, she was carried away by her vivid imagination! And I shall never forget her literally shining face and the grateful, almost overflowing eyes, when, to please her, she was assured, that her condition was not hopeless, and that it was merely a matter of time when she would learn both to hear and to speak.

The mental deficiency encountered in children suffering from some chronic organic affections or nutritional disturbances (*e. g.*, rachitis), like that associated with the aforementioned deprivation of the senses, is only relative in character. In those children the brain possesses every potentiality for normal growth and development, but remains in a state of passivity for want of prerequisite outside impressions. This is due to the fact, on the one hand, that sickly, depressed children are not at all inclined to bother with what is transpiring around them, and, on the other hand, parents justly refrain from burdening sickly children with any sort of training and education. That deficient or disturbed nutrition, *per se,* is not a potent factor in the production of amentia, can readily be proved by watching the acuity of perception of emaciated so-called marasmic babies. Nothing that bears a semblance of food or its container escapes their attention, and they show a wonderful dexterity in manipulating the nipple or bottle at a very early age.

Faulty environment and isolation, similar to deprivation of the senses, greatly retards mental elaboration owing to lack of cerebral impressions by outside influences. We can hardly expect a young child to distinguish objects it never had a chance to see or to touch; and the unfortunate child who happens to be cursed with a habitually intoxicated father, mother or both, and daily sees before him smashed heads and smashed dishes, and hears profanity on the one hand and incoherent babble on the other, is certainly ill prepared to acquire the attributes of normal mentality, and to show affection, power of imagination,

Fig. 225.—Moramentia, as a result of isolation and faulty environment.

judgment and discrimination. Faulty environment and isolation are not invariably the sad lot of the children of the poor and the degenerate. I have met with many a baby of fashion under two years of age or older, who at first impressed me as being utterly idiotic and who had remained mentally backward for several months thereafter, because of their having been secluded in some remote corner of their nurseries or huddled away under the upholstered hood of their carriages, and thus were given no opportunity to exercise their musculature or brain matter. Only too often do we see infants of the rich entrusted to the care of some inexperienced, half-baked, "white linen nurse," who considers it

her greatest achievement to keep the baby's bowels regular and who has
not the slightest conception of the importance of early mental develop-
ment. But as has previously been stated, this group of mentally back-
ward children of the poor as well as of the rich, when by Nature en-
dowed with normal brains, with marvelous celerity, they retrieve the
dormant mental faculties if placed in desirable surroundings and given
the benefit of sensible management.

Prophylaxis

The aforementioned theoretic consideration of idiocy and the allied
mental deficiencies in childhood tend greatly, I believe, to establish the
facts, first, that amentia is preventable in a large proportion of cases,
if prophylactic measures are instituted early; secondly, that under
suitable management a great many mentally deficient children can be
made useful to themselves and possibly also to the commonwealth. We
shall now endeavor to offer a few practical suggestions to accomplish
this highly desirable object in view.

One of the most essential factors in the prevention of mental debility
in the offspring is their inherent bodily and mental strength. Inherent
strength is not procurable after birth. As stated it is a consummation, an
inheritance of ancestral virility and vigor, premarital purity, conjugal
devotion, matrimonial chastity, sobriety and ideal hygiene. It can be
fostered by sensible regulation of marriage, conservative mutual selec-
tion, avoidance of consanguineous mating and prohibition of marriage
among those encumbered by chronic brain affections, grave wasting
diseases, alcoholism, drug habits and extreme poverty. Above all, in-
herent strength can be fostered by judicious management of pregnancy,
labor and the physical and mental care of the infant. Within recent
years there has been a great awakening to the importance of exacting
from those destined to procreate the race of the future that they be
free from all encumbrances, congenital as well as acquired, which tend
to embarrass their offspring in their normal development. And while
eugenics, as at present taught, carries with it a good deal of useless,
nay harmful, fiction and fetichism which veil its true object and render
it subject to ridicule and derision, there is every reason for the belief
that after the noisy agitation has ceased and thorough sifting of the
good from the bad has taken place, the world will be very much the
better for it. In the meantime, or until the lustrous millennium has
dawned upon us, it is entirely sufficient for physicians to preach practi-
cal rather than theoretic eugenics and to counsel those encumbered
by grave hereditary taints to be very cautious in the selection of their
mates, lest doubly marred heredity may intensify the degeneracy in the

offspring. All agree that those suffering from specific venereal disease, tuberculosis, malignant disease, epilepsy and insanity are not marriageable subjects, and should not be permitted to marry, unless they can show that they have remained free from any traces of these affections for a number of years. But it is not in the province of the physician to join the eugenists in their hunt for "desirable types" of man- or womanhood, even were such types at all desirable. "What the eugenists set up as desirable types," says A. C. Jacobson, "strikes many of us as merely smug, unctuously respectable and commonplace paragons. If the eugenists had their way and succeeded in peopling the world with a race of disgustingly normal beings, standardized to the Philistian scale which the intellectual plebeians who are so warmly drawn towards the eugenic camp seem determined to devise, life would be drab and jejune indeed. Happily such a consummation can never be, for which the gods be thanked. Anything approaching real control of the race after the plan of these fanatical breeders is a phantasy * * * since haphazard 'scrub-breeding' has gone on in the human family so long that pure strains with definite character units are practically unknown. Hence, who is the fit? What is fitness"? Physicians can do most good by judicious management of pregnancy and labor, and the rearing of the child, more especially during its first few years of life. As has already been stated, after impregnation the destiny of the offspring is partially or wholly dependent upon the physical and mental welfare of the mother. "But even if it be proved —it has not yet been proved—that the conditions of life in the nine months before birth have no influence either for good or ill upon hereditary maladies and deformities, even then there remains much to be done in antenatal hygiene, for there cannot be the slightest doubt that many morbid influences come to play upon the body of the infant in the womb and that some at least of them may be prevented or their results cured" (Ballantyne). No definite statistics have thus far been adduced to show the degree or extent of the beneficial influence of antenatal hygiene upon the mentality of the offspring, but some approximate estimate can be obtained by analogy, when we compare the weight and physical power of resistance of babies born under favorable conditions with those born of mothers who up to the last moment of pregnancy were exposed to hardship and struggle for existence. Thus, Pinard gives as the average weight of babies of women, who worked up to the time of delivery, about 6½ pounds, while for those born of women, who had a short respite from hard work before delivery, 7¼ pounds. Bordé found the average weight of babies of Italian women, who worked up to the delivery, to be 6¼ pounds, while of those, who had rested a few

weeks before delivery, about 7 pounds. More recently S. Peller reported his findings among Austrian women. His material was drawn from two sources, a sanatorium for women of means with 612 patients, and a large clinic for poor women (under the direction of L. Teleky) with 4,875 cases. He found that the first born male babies of well-to-do women averaged about 4 ounces heavier and the female babies about 3 ounces heavier than the babies of poor women who worked up to confinement. Moreover, in a comparison of the first born children of hospital women with those of the women coming to the hospital just before confinement, the babies of the former are shown to average about 4 ounces heavier than those of the latter group.

With these observations in view the importance of antenatal hygiene for the betterment of the race of the future becomes self-evident. The prospective mother should be placed in a wholesome environment and proper hygienic surroundings. Her diet should be liberal, her living rooms spacious, and airy, and her association cheerful. Wherever possible, she should be free from the anxieties of earning a livelihood or the pompous frivolities of wanton society. The boundless extravagancies of extreme wealth and the awful misery of extreme poverty, both alike, sap the vital forces of the mothers as well as of their offspring. The State, if need be, should provide for the poor expectant mother at least a few weeks' respite from hard work previous to delivery and also thereafter. We must allay the anxiety of the primapara by assuring her that pregnancy and parturition are physiologic, normal processes, under proper management devoid of perilous complications and sequelæ; and the multipara should be impressed with the fact that miscarriages and attempted abortions are dangerous experiments, one tending to interfere with normal development of the offspring that are to come later (by leaving the uterus in a more or less permanently diseased state), the other actually injuring the embryo or fetus during the process of growth and development.

Next to antenatal hygiene the judicious management of labor serves as the most important means in the prevention of amentia in the offspring. Judging by the appalling number[1] of cases of paralytic amentia following traumatism during birth, and considering the fact that the cases recorded form but an infinitesimal portion[2] of the innumerable cases that never see light after delivery or survive the injuries sustained but a few days or weeks, there must be something very seriously

---

[1] Lapage reports 25 out of 96 cases of amentia under his observation; Still 26 out of 135; my own records show 30 cases out of 119.

[2] In Philadelphia, for example, out of 39,975 births during the year of 1911, 2,131 were stillborn; and according to the annual report of Miss Julia C. Lathrop, out of 300,000 infants under one year who succumbed during 1911, about 30 per cent did not live to complete the first month of life as a result of prenatal conditions or of injury and accident during birth!

wrong with the way midwifery is being practiced even in the civilized parts of the world. Due allowance, of course, must be made to the fact that in this country the women who furnish the greatest number of births are of foreign birth and bringing up, and owing to mistaken prudery still cling to the custom of their mothers and depend upon ignorant midwives for the performance of the vital function of obstetrician. But it is high time that each state or the Federal government should put a limit to this much abused ''personal liberty'' clause and insist upon only licensed midwives (after practical examinations) being permitted to practice obstetrics, in order to safeguard the future welfare of the children as well as of the mothers, not to speak of the economic benefits to their respective communities. Medical men also ought to awaken to the gravity of the situation, and, on the one hand, refrain from the hasty application of instruments in ordinary cases, and, on the other hand, in difficult labors not to hesitate to invoke the assistance of competent obstetricians, who through skillful manipulation might possibly be able to prevent cranial injury, asphyxia, etc. in the infant, which so frequently lead to cerebral diplegia with amentia. Pituitary extract, the most recent addition to the obstetrician's armamentarium, which in appropriate cases seems to exert an almost magical effect upon inertia uteri, will undoubtedly greatly help to dispense with instrumental delivery and thus to diminish the number of aments due to this cause.

As regards the postnatal care of the infant in the prevention of mental deficiency, let me urge upon physicians ever to remember that their sphere of usefulness does not end with the perfunctory manipulation of the stethescope or thermometer, nor even with the punctilious elaboration of food-formulas, diet lists or recipes. The mentality, *das Sinnesleben,* (the mind activity, or the senses) of the child should enlist as much of our deliberative attention as its physical condition, more especially when there is a tainted family history or an environment that is conducive towards a morbid mentality. And while it surely is the physician's paramount duty to safeguard the physical welfare of the child, it is no less important for us to guide its mental destiny. Indeed, strength of mind quite often compensates for weakness of the body, while strength of body only very rarely, if ever, compensates for weakness of mind.

In the mental training of infants we usually meet with two extremes. One class of mothers keeps its infants in a state of *noli me tangere,* hidden in the remotest corner of the boudoir, lest it be bewitched by an ''evil eye,'' or, as they say, unhinged by the premature sensitization of the brain; the other class of mothers injudiciously strains its infants'

cerebral functions to the breaking point, makes them the central figures of attraction of their household, teaches them to sing and to recite, to read and to write at a very tender age, when they are barely out of their bottling period. These two extremes in mental training should resolutely be discountenanced, and the happy medium chosen. The infantile brain, like the potter's clay, needs moulding while it is fresh and pliable, but it must be mastered skillfully and gently to avoid exhaustion of the brain cells or their disarrangement. The mental training should begin when the infant is about three months old. He should be picked up a few times a day, put on the lap and supported with the forearm, and shown a few lustrous things to stimulate his power of vision and attention. Gradually some object should be put in his hands to train him to grasp. As he gets a little older, he should now and then, for a few minutes at a time, and properly supported, be sat up in his carriage or bed, and allowed, as it were, to make a general survey of the beautiful world and the grand things that help to make it so. At six months of age, if strong enough, he should be put in a baby chair, given a few harmless little toys to play with, and be permitted to be accosted by some intimate friends of the family, in order to get the child accustomed to distinguish strange faces. Some few months later, he should be gradually taught to perform some simple baby tricks, such as clapping hands, and the like. In suggesting these procedures I do not at all intend to convey the idea that every kin and friend of the family should be invited to exhaust their ingenuity to devise means to entertain the baby. Quite the contrary, we must ever bear in mind that an infant is easily fatigued, irritated and indisposed, and hence should not be overtaxed even by the simplest methods of training. This holds true also of older children, for many a supposedly nervous, naughty, intractable, listless or morose child, on careful inquiry, is found to be suffering from the effects of overtaxation of its mind by injudicious entertaining, or training and education.

On a previous occasion attention has been directed to the serious consequences, in the way of mental affections, not rarely following febrile diseases of childhood. Here also it is in the physician's province to educate the public that measles is not a "children's ailment that every child must go through," that whooping cough is not "harmless and makes children fat thereafter," and that "scarlet rashes" are "of no consequence and the result of a spoiled stomach or teething." And the sooner the people will appreciate that grave danger lurks even in the most benignly appearing attacks of infectious diseases, the sooner will the mental deficiencies arising from this cause dwindle down to insignificance.

Active Treatment

We can now pass on to the treatment of amentia, and since this chapter is intended for the discussion of mental deficiencies in infants and children under school age only, we propose to speak only of therapeutic measures, (hygienic, pedagogic, physical, medicinal and surgical) as they are applicable chiefly in the management of aments under five years of age.

Hygiene

An ample supply of fresh air and good food, bodily cleanliness and proper clothing are essential prerequisites; in many respects more so in the care of aments than in normal children. Owing to the tendency of mentally deficient children to contract tuberculosis and the frequency of respiratory embarrassment as a result of nasal disease or deformity, they should be kept outdoors most of the day and in thoroughly ventilated rooms during the night or inclemency of the weather. This fact should strongly be impressed upon those who take care of these children, since most mothers are apt to mistake the cause for the effect and attribute the difficult breathing and nasopharyngeal catarrh to "catching cold" in the street.

Several precautions have to be taken in feeding mentally backward infants. Some of them owing to their voracious appetite and lack of prehension of the sense of heat or proportion, are apt either to burn their mouths with hot food or swallow big morsels, and thus permanently impair their powers of digestion. In the majority of cases, therefore, it is required to prepare and subdivide the food properly, just ready for consumption. Others again because of an imperfect sense of taste and inability to manipulate the tongue, often refuse food, especially solids, and have to be fed with small quantities of food at frequent intervals. Owing to nasal obstruction, either as the result of adenoids, nasal deformities, or general debility, some congenital idiots experience difficulty to nurse at the breast; hence, it is often necessary to pump off the breast milk and to feed the baby either through a bottle and small nipple or by means of a spoon. As soon as possible, let us say from the eighth month on, mentally backward infants should be put on a mixed diet, in order to prevent rachitis or scurvy. We usually begin with small quantities of fresh fruit juice, beef juice, strained vegetable soups, coddled eggs, cereals with milk, stewed fruit and vegetable purée, gradually increasing the quantity of food as they get older, so that at the age of three or four years they can be put on the following dietary:

On rising in the morning, 4 ounces of milk, preferably boiled.

One hour later, about 8 a.m., ½ ounce of orange, pineapple, or grapefruit juice; 4 to 6 ounces of well-cooked cereal in milk and a little butter, *e.g.*, oatmeal, farina, sago, rice, tapioca, cream of wheat or arrowroot; milk toast, and a coddled or poached egg.

At about 12 m., 4 ounces of broth with some cereal or toasted bread; 2 ounces of vegetables (potatoes, carrots, spinach, cauliflower, beans, peas, etc.) well cooked and finely mashed; ½ ounce of finely chopped scraped beef, chicken or lamb chop, or boiled or broiled white fish. If the child is still hungry we may add a slice or two of stale bread and butter, divided into small pieces.

At 4 p.m. one cup of boiled milk with a few biscuits with jelly or butter; or a ripe banana.

At 6 p.m., one coddled egg, bread and butter or jelly, and 4 ounces of boiled milk; or cereal pudding or custard, bread and jam or treacle; or a cup of cocoa with toasted bread thrown in and a small portion of stewed fruit.

Water should be given between meals.

Before and after each meal the child's hands and face should be thoroughly washed as a routine procedure, which may aid also in teaching the child cleanly habits.

The training of cleanliness is very essential to the child's future welfare, since it not only serves to make it more presentable to those coming in contact with it, but, which is by far more important, it acts as the most efficient preventative of divers local and constitutional infections. In addition to frequent local cleansing of the body as necessity arises, it should receive a tub bath daily, preferably in the evening, in the same manner as normal children. Regardless of the mental condition of the child, every effort should be made to train him to respond to Nature's calls. From six months of age on he should be put on a nursery chair at regular intervals, at first every two hours and later every three or four hours. If his bowels do not move spontaneously after he has been sitting on the chair for several minutes, the infant should be trained to press by inserting into the rectum a small soap stick or glycerine suppository. After persistent training even the idiot will gradually learn to understand what is expected of him, when placed upon the nursery chair, and in time he will of his own accord announce his desire to urinate or defecate. Aments more so than normal children, should receive more care as regards changing of diapers and keeping the mouth, nose and eyes clean, in view of the fact that, as a rule, they are less sensitive to pain and annoyance, and hence are less apt to complain when those portions of the body are in an irritated state. Moreover, special attention should be paid to apparently the simplest kinds of cutaneous eruptions as these often serve as portals of entry to systemic infections.

Mentally deficient infants, especially if delicate and thin, should be very warmly dressed. They may suffer greatly from the effects of cold and yet fail to appreciate it, owing to dulness of sensibility. Chilblains and frost bites are quite common among them and general circulatory disturbances are frequently encountered, especially in Mongolians and cretins. Flannel and silk underwear should be given preference to cotton or flannelette. During the cold season special protection should be accorded to the hands, feet and ears, and very delicate infants may preferably be kept indoors, in well ventilated rooms, particularly if they show a marked tendency to congestion of the nasopharyngeal mucous membrane.

"Incentive" Training, and Physical Therapeutic Measures

The main object of systematic training in amentia is to render the mentally backward child capable to help himself in the care of his body, to look out for his health and comfort, and later to learn some simple occupation to earn a livelihood. This requires first of all the ability to exercise the voluntary musculature. Since in a great many aments several groups of muscles are either atrophied and incapacitated from disuse or actually paralyzed, we must endeavor to establish or reestablish their functions by passive motion, massage, hydrotherapy, electricity and active exercise. These procedures must be continued uninterruptedly daily for months and sometimes for several years, notwithstanding their seeming futility. In the end we are usually amply rewarded by success for our patience and perseverance. Even in the paralytic it has repeatedly been found that the function of the diseased cerebral area has been taken up by the corresponding healthy structures of the brain; and as has been shown by Vitzow, Pfitzner, Merk, Cattani, Klebs and others, in exceptional cases actual regeneration of nerve tissue occurs where its injury is not very pronounced. The physical treatment of the affected limbs should begin as soon as their weakness or paralysis has been determined regardless of whether or not reaction of degeneration has supposedly taken place. For it is by far safer to err in the direction of overtreatment than undertreatment. Let me illustrate this point by a case under my observation. It concerns a three-year-old boy who received severe cranial injuries during instrumental delivery. The abrasions from the blades of the forceps were still visible six weeks after birth, when the child came under my care. There was at the time distinct paralysis of the face, of both upper extremities and of the right leg. A few days before, the baby was seen by a noted neurologist who thought there was no hope of his ever recovering, and discouraged further

treatment. The case did look hopeless, yet the parents failed to reason as disinterestedly as the learned doctor did, and I also agreed with them that the baby ought to be given a chance to fight for his own. The facial paralysis proved peripheral in character and the paralysis of the left arm was of the Duchenne-Erb type; both disappeared under massage, electricity and patience, while the right hemiplegia has improved so much that the boy is able to walk about with ease and to participate in all sorts of children's games. Moreover, his mentality seems perfectly normal. I may add, by the way, that his cranial circumference measures about 22 inches, and by its shape and consistency gives the impression of a macrocephalus accompanying hypertrophy of the brain.

There are a few practical points to keep in mind in the application of massage, electricity and hydrotherapy. The massage movements should consist of stroking, friction, kneading, light pinching, tapping and rhythmic vibration. The duration of each treatment should vary from a few minutes in the beginning up to a quarter of an hour after the child has become used to the manipulations. This should be followed by passive motion of a few minutes' duration. The massage should be gentle, preferably by means of talcum powder, since it allows the hands to glide smoothly over the body surface.

Electricity should be administered from ten to twenty minutes at a sitting, either daily or every other day, using the mildest current that will cause muscular contraction without undue pain. The galvanic and faradic currents, alternated with the sinusoidal, answer the purpose well. If single muscles or muscle groups are affected, the sponge electrodes are to be applied near or at the points of origin and insertion of the muscles, while if whole extremities are involved, we apply a large flat sponge electrode, well moistened in warm salt water, on the spine and stroke the affected muscles with a small electrode.

Hydrotherapy is particularly useful in amentia associated with muscular rigidity and general cerebral irritability. A warm (98° F. to 101° F.) tub bath, of from five to ten minutes' duration, should be given once or twice a day, and while the child is in the tub its limbs should be gently rubbed with a rough flannel and if possible extended and moved in all directions.

Simultaneously with the application of these therapeutic procedures, we employ systematic training of the voluntary musculature and of the special senses, in order to foster the physical and mental development of the backward child. In pursuing this course of treatment we must as closely as possible follow the successive steps taken by Nature in the unfolding of the human intellectual faculties, and avail

ourselves of the child's natural instincts to assist us in our efforts. In our study of the normal baby we have noted that immediately after birth he is endowed with the instinct of suckling, or "fishing" for food, and to cry when hungry or thirsty. This instinct is as strongly developed in the idiot as in the normal child. Now, then, since the struggle for food, for self-preservation, forms the ever and everywhere dominating and propelling force of evolution in the animal kingdom of the entire universe, and has formed the most vital incentive even in primitive man to devise ways and means for its sustenance and perpetuation, I believe that the irresistible force to quench thirst and to appease hunger ought to be sufficiently powerful to awaken even the total idiot from his mental torpor, and to induce him, as it were, to struggle for his existence. Indeed, the longer I practice this *incentive* method of training of the mentally defective infant, the more convinced I am of its superiority over every other method of training in vogue. We make use of his desire for food to teach him how to look, how to listen, how to pay attention, how to grasp, how to imitate personally and with objects, how to walk and how to talk—all in the order in which the normal baby acquires these faculties, except, of course, at greatly delayed periods as compared with the age of the normal baby. The sooner the training is begun, the more promising are the results, principally because in amentia of long standing the brain cells usually entirely lose their regenerative quality. This fact should strongly be impressed upon the unfortunate parents who rarely note any mental deficiency in their infants, and if they do, are often led to believe that they will "outgrow their weakness when they get to be seven years old."

As amentia is very readily recognizable in an infant about six months of age, we proceed with the training in the following manner:

1. If it is a nursing baby, he is put on the lap facing the mother or wet-nurse, and, after exposing the breast, the baby's hands are brought in contact with it and manipulated so as to make them grasp it. This is repeated for a few minutes before each nursing. We next bring the baby near the breast nipple and squirt a little milk in his mouth. As the baby puckers his lips to grasp the breast nipple, we promptly pull the child back, so as to force him to struggle to get a good hold of it. This also is repeated for a few minutes. If it is a bottle baby, we perform the same maneuvers with the bottle and nipple. As he gradually learns to recognize the bottle, we next endeavor to train the baby to follow the course of the bottle by slowly moving it before his eyes in all directions, before allowing him to get hold of the nipple as it comes near his mouth. It may take several

days, weeks or even months to accomplish this trick, but patience and perseverance are the keynote to success in the training of the mentally defective.

2. After the baby has acquired the power of grasping the bottle, we place the child in a semirecumbent posture with the head resting on a small pillow, put the bottle in his hands, and as he is about to start to suck the nipple, we slowly pull the bottle backwards and continue to do so while the child is making every effort to bring the bottle to his mouth, and by hanging on to the bottle lifts himself from the recumbent to a sitting posture. This up-and down-movement is exceedingly useful to strengthen the arm- and spinal muscles and to train the child to raise himself from a recumbent posture. Of course, these exercises must be continued for several minutes before each feeding.

3. Like normal babies, aments also should receive a mixed diet when they reach eight or nine months of age. Most of them, as a rule, can be readily induced to take eggs and fruit, which are most excellent bone and brain builders. We hold the egg in front of him and feed him with a spoon at short intervals, making him wait for the next spoonful until he shows anxiety to get it. If a white egg does not attract his attention, we color it red or blue, and move it in different directions to teach him to follow objects. Similarly, hold a red apple in front of him, scrape some of it, and feed him at short intervals; bring the fruit near his nose and let him learn to perceive its odor. Some idiots have a highly sensitive sense of smell, and by using attractive odors as a bait, they can be induced to awaken from their apathy and to respond better to the systematic training. Next, place the child near the side of the crib and put his hands on the upper cross-bar, and while you hold the apple at a short distance above his head, with one hand, help the child to lift himself from his position with the other hand; as he accomplishes it, let him have some of the apple, (orange or peppermint stick), and let him go through the same performances again and again to earn some more of it.

4. By means of a rod and cord, suspend a red apple or orange in front of the baby, and let it hang there for a little while. If he remains passive, bring the apple near him and let him grasp, smell and taste it; if he is now attracted by it, swing the apple to and fro and encourage him to follow it with the hands and grasp it. Repeat the exercise several times and let him have some of the apple for each successful effort. Toys may be used instead of eatables where the child shows preference for the former. By continuing these exercises

the child gradually gains considerable power of attention and muscular coordination.

5. Aments should early be taught to feed themselves. Sit him in a baby chair, if need be well supported with pillows, and place the food before him. Give him a taste of it, and if he is good and hungry, he will "fish" for more. In this event, if the food is solid (*e. g.*, a zwieback), put it in his hand and guide him repeatedly to bring it to his mouth. It is usually a very difficult task, to teach mentally deficient children to feed themselves, but hunger and persistent training will accomplish it in the end.

6. To teach him to stand, we place him against the side of the bed with his arms crossing the top bar, and feed him with a spoon in such a manner that he is forced to raise his head to receive the food. In the beginning, it is usually required to support his back to keep him from falling. As he learns to stand, put him in a softly padded walker, the top of which snugly surrounds his waist. Keep the dish of food in front of him, give him a mouthful of it and take a short step backward; let him follow you (which in the beginning may call for your assistance); give him another mouthful and let him again push forward. Repeat this a few times a day, at first only for a very short time, in order not to tire him. A doll carriage may occasionally suffice as a support instead of a walker, especially after the child has partly learned to walk, and only needs additional exercise.

7. Gradually train him to walk without any support. This is best accomplished first by standing him against a wall, and while facing him, let him grasp one of your index fingers, and follow you while you take single steps backward. Later extend to him a cane or rod instead of your fingers. If he hesitates to follow you, use some fruit, sugar or candy as an incentive, which you hold in front of him and reward him with for successful efforts. Or put his food on a low table, direct his attention to it and lead him towards it. In time he will go after his food without being led.

8. All the while the child is receiving instructions keep on telling him what you are doing and what you wish him to do, regardless of whether or not he understands you. Gradually he will learn to understand at least part of what you are telling him. Use single words instead of sentences, *e. g.*, eat, drink, walk, etc., and repeat the words in a firm tone of voice, in order to make a lasting impression upon the auditory center.

9. After he has learned the different exercises, you can begin to interest him in drills, tricks, and games as practiced in modern kindergartens. Almost all mentally deficient children are charmed by

music; it is therefore of great advantage to make use of harmonious strains of the piano to arouse the dull child from his slumber and to soothe the discordant impulses of the agitated child. Different strains of music should be used for different sets of actions, in order to train the child's auditory apparatus to connect the particular melody with the particular act he is to perform; in other words, one and the same melody with his meals, another one when he marches, a third when he plays a certain game, etc. Music should be employed also in training him to speak. Thus, while playing the piano sit the child in front of you, attract his attention to your mouth, and, with a tone of voice corresponding to the strains of music, keep on repeating single syllables or words, e. g., ba-ba for one melody, la-la for another, ta-ta for a third and so forth, gradually lengthening the syllables into whole words. In speech, as in other exercises, its accomplishment is often facilitated by using food as an incentive, i. e., give him a piece of candy every time he makes an earnest effort to pronounce certain syllables or words.

10. Imitation is the mother of experience. Teach him to imitate your personal movements, such as kneeling, sitting, standing, opening and closing of the mouth or hand, throwing a ball or catching it, and similar exercises.

11. Sit the child near a table facing you; spread out in front of him some candy or some other eatable he is fond of, and let him taste some of it. If he likes it, he will surely look for some more of it. Now cover the remaining pieces of candy with a strip of paper, leaving part of it exposed. If he shows ability to remove the paper and to help himself to the candy, put the latter in a little box, first without a cover and then with a transparent cover. Now teach him to uncover the box, and, if he succeeds doing it, reward him with a piece of candy. Next put the candy in a more complicated contrivance, and the more ability he shows to help himself, the more difficult should it be made for him to find the thing he is looking for. By repeated training he will gradually learn to help himself in many other respects.

12. Place the child in front of a step ladder and put the candy on one of the rungs and encourage him to reach for it. Of course, at first he will need your help. As he ascends the ladder, hand him a piece of candy. Repeat this maneuver again and again, and as he succeeds in getting there, elevate the box to a higher level. In a similar manner reverse the performance, i. e., make him descend in order to reach the box. As he learns to accomplish this with ease,

teach him to climb stairs, first by supporting himself with the hands and later by doing it without support.

The performances, of course, can be multiplied almost *ad infinitum*. But there are two essential ideas ever to be kept in view in the training of aments—namely, first, no coercion or force is to be applied; second, no time and energy should be wasted on exercises which are not absolutely indispensable to his welfare. If we succeed in training a deficient child under five years of age, to be clean, to feed himself, to walk, to understand words spoken to him and, possibly, to make himself understood, even if only by single words, enough indeed will have been accomplished. By opening up the avenues of approach to the dormant, deficient infantile brain, the brain itself will spontaneously evolve its resourcefulness, round out the experience, and receive new impressions.

In suggesting the aforementioned exercises I presume, of course, that we are not dealing with total idiocy accompanying extreme degrees of hydrocephalus, microcephalus, diplegia, etc., or amaurotic family idiocy. In these cases no amount of conscientious training ever will bear fruit in restoring degenerated brain tissues to normal function. Moreover, the span of life of these unfortunates measures but a few years. On the other hand, when confronted by a mentally deficient child who is free from gross cerebral lesions and shows some response to outside influences—for example, a child of two years responds to the mental tests for a normal baby of six months—painstaking and persistent training will most assuredly bring forth very gratifying results, even though in the beginning nothing but failure will seem to crown our efforts. The late Edouard Séguin, one of the early pioneers in the training of mental defectives, when once asked why he kept on repeating the same movements a hundred times a day, replied because the child does not make them right ninety-nine times. This was the secret of his phenomenal success. And it is essential to impress upon the parents that unless they themselves are endowed with an ample supply of patience, tact and perseverance, to keep on teaching their child the same thing for days, weeks and months, this work should be intrusted to some one who possesses these qualities, or no great achievements need be expected.

Finally, in training the weak-minded it is well to remember that the unfortunate baby is not to be blamed for his failure promptly to apprehend and to copy the apparently simplest rudiments of thought and action. He is heartily to be pitied rather than disdained; and as the great majority of aments are providentially blessed with a con-

tent and joyful disposition, we might as well refrain from shattering their peace of mind by undue harshness or rough handling.

Medicinal Treatment

When discussing cretinism, attention has been directed to the marvelous physical and mental transformation occurring on the administration of thyroid extract in amentia due to thyroid insufficiency. Wherever the cause of amentia was uncertain, I made it a rule, to give the child the benefit of a few weeks' thyroid treatment in order to determine, whether or not the thyroid was at fault. Moreover, for the last two or three years I believe to have found it of advantage to supplement the thyroid medication by the extracts of parathyroid, thymus, and pineal and pituitary glands, in accordance with the established fact that whenever the thyroid is affected, the functions of the other glands are also more or less impaired. In one case, particularly, the effect of the combined glandular medication was singularly striking. It concerned a six-year-old boy who for three years had been treated with thyroid extract by several eminent clinicians. When I first saw him he measured 35 inches in height and weighed 41 pounds. His voice was husky, and he could pronounce but a few words in a draggy, staccato sort of fashion. His face and lips were edematous, and his tongue protruded slightly. When led by the hand he was able leisurely and awkwardly to move along, but if left alone he was barely able to take a few steps without stumbling. I put him on the aforementioned glandular extract compound, and he gained 1½ inches in height, 3 pounds in weight, and a great deal in intelligence. He became so active that the mother experienced considerable difficulty to "restrain him from following the boys in the gutter." As, until he came under my observation, he had regularly been receiving from 2 to 5 grains of thyroid extract daily without appreciable benefit, I could not help but believe that the marked improvement in his condition was due solely to the addition of the parathyroid, thymus, pineal and pituitary extracts. The mode of administration of the glandular extracts varies somewhat with the age of the child and the degree and duration of the affection. In congenital cretinism I order 1 grain of thyroid powder twice a day, to a bottle-fed baby, and only half the quantity to a breast-fed infant, having observed that the latter develops the manifestations of cretinism more slowly than the former, owing probably to the fact that the breast baby in the first few months of life receives some thyroid through the mother's milk. If the case fails to show improvement in about four weeks, I begin to alternate with ¼ grain each of

parathyroid, thymus, pineal and pituitary extract. This represents the usual dosage for an infant up to one year of age. Older children should receive $\frac{1}{2}$ grain of the thyroid powder and $\frac{1}{4}$ grain of each of the other glandular substances for every additional year of their respective ages up to five years. The dosage in tablet-form is about twice as large as that of the powder. After considerable improvement has taken place in the child's general development, the dose of the thyroid or the compound should gradually be reduced to once a day, once every alternate day, and once every third day. Where organotherapy gives rise to cardiac palpitation, undue restlessness, or gastric irritability, the treatment should temporarily be suspended until the toxic symptoms have disappeared, when the medication should be resumed in smaller quantities, and gradually increased.

There are several other medicinal preparations which have to be resorted to in the management of the different forms of amentia. Regardless of cause, it is often judicious to place an ament on a thorough antisyphilitic treatment, more especially, of course, when the Wassermann reaction is positive or there are reasons to suspect syphilis either from the history of the case or appearance of the patient. Opinions are still at variance as regards the advisability of employing neosalvarsan in the treatment of syphilitic amentia in children, and I am inclined to give preference to the mixed iodide and mercury treatment, unless there be special need for hasty action, e. g., syphilitic hydrocephalus with marked intracranial pressure. If neosalvarsan is indicated it should be administered either intravenously or intramuscularly. The usual dose and mode of administration is fully given when discussing the treatment of syphilis (see p. 494). The effect of the neosalvarsan should be controlled by the Wassermann reaction, a second dose being administered after two weeks if necessity arises.

The iodides are indicated even in the absence of a syphilitic taint, acting as they do as powerful alteratives and eliminants of divers systemic poisons. One grain of the sodium iodide, twice daily, for every year of the child's age, is ample for ordinary purposes. The syrup iodide of iron in 10 to 20 drop doses may be alternated with the sodium iodide, or some of the newer iodide preparations may be used instead. To obtain results the iodide should be continued for several months, with occasional intermissions of short duration, in order to avoid gastric irritation. It is often found very beneficial to combine the syrup iodide of iron with cod liver oil and the syrup of lime hypophosphites, more especially where rachitis complicates the amentia. General tonics and appetizers are almost always indicated, for we hardly could make proper

use of our *incentive* method of training, when the incentive, hunger, is lacking. Small doses of the tincture of nux vomica and cinchona compound, in orange syrup, before meals, and dilute hydrochloric acid and essence of pepsin after meals, act exceedingly well both as tonics and digestants and should be prescribed as necessity arises. In paralysis or general muscular debility it is often advisable to administer strychnine by mouth or even hypodermically. One-three-hundredth of a grain for every year of the child's age up to five years will ordinarily suffice in cases of moderate severity. The dose may be repeated twice or three times a day. Sometimes sedatives are indicated, and I have found that small, frequently repeated doses of codeine, dionin or heroin, act very much more promptly in relieving attacks of twitching, extreme restlessness and insomnia, than the bromides or other hypnotics. Of course, in epilepsy, the bromides or luminal are indispensable, and instead of, as is usually advised, giving small, gradually increased quantities of bromide, I have found it very much more profitable to start with large doses, and to reduce them, after the periodic attacks have been arrested. Thus, for every year of the child's age I give 1 grain each of potassium, sodium and strontium bromide, three times a day, and continue the same until I have succeeded in arresting the usual fit for several months. Then the dosage may slowly be reduced if it is found that the child is too drowsy or signs of bromism make their appearance. I prefer to combine the bromides with small doses of Fowler's solution and the mixture of rhubarb and soda, the arsenic seemingly preventing bromism while at the same time acting as a nerve tonic, and the rhubarb and soda serving to subdue the undue gastric irritation. In habitual constipation which is the rule in mentally deficient babies, $\frac{1}{2}$ grain of phenolphthalein or 10 drops of aromatic fluid extract of cascara sagrada, for every year of the child's age, or liquid petrolatum in teaspoonful doses will be found efficient, particularly if the movement be started with a small injection of warm soap water or glycerine suppository. In very young infants milk of magnesia (1 or 2 teaspoonfuls, best mixed with the entire 24 hours' quantity of milk) usually answers the purpose. Special attention should be paid to the nasopharynx. Where adenoids exist and greatly interfere with respiration, they should be promptly removed, otherwise they can be kept from doing much harm by keeping the nasopharynx clean with Dobell's solution and adrenalin (1:1000), equal parts, and the instillation into each nostril of a few drops of a 10 per cent solution of argyrol, solargentum, or the like, twice a week, until the inflammation and hypertrophy of the adenoid tissue has considerably subsided. Intercurrent diseases, of course, must be treated according to indications in the same manner in aments as in normal children, ex-

cept that greater attention must be paid to the prevention of passive, hypostatic pneumonia, and even to trifling ailments which ordinarily are entirely free from complications in normally developed children.

Surgical Treatment

Surgery as an aid in the cure of idiocy and the allied mental deficiencies has been resorted to especially during the last two decades. The results obtained, however, are far from being satisfactory, except in cases of paralytic amentia due to cerebral compression, where early decompression has not rarely brought about complete regeneration of the brain tissues involved and *restitutio ad integrum.* Harvey Cushing has performed quite a number of craniectomies on the newborn to relieve cerebral compression resulting from intracranial hemorrhage during birth (which, as previously mentioned, forms the cause of amentia in about 30 per cent of the cases on record), and is of the opinion that with proper regard of hemostasis and careful avoidance of undue exposure, the newborn will stand a cranial operation well, its life will often be saved, and in many instances develop normally. Roswell Park maintained that where a reasonable integrity of brain structure can be assumed, there was no reason why craniectomy or decompression should not be given an opportunity in imbecility and psychic disturbances, in order to relieve pressure and permit more normal development. And William Sharpe and H. F. Farrell claim* very good results from cranial decompression in cases of spastic paralysis (with mental deficiency) of hemiplegic, paraplegic, and diplegic type with a *definite history of difficult labor with or without the use of instruments, in which on ophthalmoscopic examination signs of intracranial pressure are shown in the dilated retinal veins, and a blurring and haziness of the optic discs, especially of their nasal halves.* In these cases they perform a large, right subtemporal decompression, and if the intracranial pressure remains high, they perform a left subtemporal decompression the following month. The after-treatment which is of very great importance, consists in the correction of deformities by tendon lengthening and stretching of the contracted muscles: the maintenance of the corrected positions by the employment of especially adapted and properly fitting braces, and skilled massage in conjunction with short applications of galvanism and faradism. A careful, systematic course of muscle training is carried out daily. Sharpe and Farrell claim marked improvement not only in the spasticity but in the mental condition of the patients as well, so much so

---

*In several cases under observation the results proved negative.

that they are able "to receive the cooperation of the patient in the carrying out of the after-treatment." Of course, the earlier, the decompression is performed the greater the opportunity and facility for the compressed brain structures to adjust themselves in their normal relations and to regain their normal functions.

As has already been stated the results from operative interference in the other forms of amentia are, to say the least, but temporary. Lannelogue's craniectomy for the relief of microcephalus, which at first was hailed as a great success, soon proved a total failure and has rightly been abandoned even by its most enthusiastic exponents. Several operations have recently been proposed for the cure of hydrocephalus, but it is too early to arrive at correct conclusions regarding the improvement in the child's mentality and its permanency. Of these operations I may mention G. Anton's efforts to relieve intracranial pressure by puncture of the corpus callosum, and Irving S. Haynes' method of treating hydrocephalus by cisterna-sinus drainage.

In recommending operative interference for the relief of congenital or acquired physical defects complicating amentia and for the eventual restoration to normal mentality, considerable conservatism, of course, should be exercised in the proper selection of the cases. But, whenever in our judgment the case in question is entirely hopeless if left alone, and there is the remotest chance, through surgical interference to relieve the idiot of his lifelong misery, we should not at all hesitate to recommend surgical treatment, notwithstanding the accompanying appalling mortality. However, before resorting to surgical intervention, the mental defective should for a reasonable time be given the benefit of some of the other therapeutic measures here suggested.

Prognosis

Do what you will, even under most scrupulous application of all of the aforementioned preventive and curative measures, amentia, either of prenatal, natal, or postnatal origin, will persist and exist as long as man will inhabit this world. Fortunately the majority of cases of genuine congenital idiots are usually short lived. This is true especially of the congenital hydrocephalic, paralytic (associated with porencephaly and cerebral sclerosis), Mongolian with congenital heart disease, and the amaurotic. The great majority of them succumb during early infancy or childhood, either to general debility or to intercurrent diseases, more particularly pneumonia and tuberculosis. The pneumonia is usually of the hypostatic variety that readily supervenes after trifling ailments which in any way tend to depress the vitality of these weaklings. The prevalence of tuberculosis among them can

often be traced to an hereditary disposition, superinduced by the ever present nasopharyngitis and adenoids, or to direct infection of the alimentary or respiratory tract as a result of the extremely unhygienic habits of the majority of low grade aments. As is well known, idiots delight in rolling in filth and chewing on anything and everything picked from dirty floors and streets, and it is quite reasonable to suppose that these things harbor a multitude of pathogenic microorganisms, the tubercle bacillus among them. On rare occasions we meet with aments of very robust constitution, who are not susceptible to the ordinary children's diseases, and often, almost for spite, surmount violent attacks of exanthemata, gastroenteritis and the like, even if left alone without proper hygienic care, suitable feeding or medication. This is often true of the microcephalic idiot with the miniature brain and head (see p. 707), and the postnatal cretin.

The mortality in feeble-mindedness of postnatal origin, more especially in children who receive good care and treatment, as a rule, is not much higher than in normal children, provided they remain free from convulsive seizures and are able to be around and about. As regards the future mental progress of aments, each case must be judged individually. As a rule, however, mentally deficient children who at about three years of age are able to make more or less free use of their extremities, in the course of time are amenable to proper training. Some of them as they grow older can be made useful by teaching them light outdoor occupations, such as gardening, or to assist in farming, others by learning to help along in different trades, e. g., basket making, carpet laying, carpentry, and others again by doing errands, etc. Under these circumstances, more particularly, since errors in the diagnosis of the exact type of amentia dealt with are not at all uncommon even with the most experienced observers, it is hardly just or expedient to declare a case of feeble-mindedness unimprovable without giving it a fair test by way of physical and mental training, or, possibly medical or surgical treatment, whenever there is reason to believe that these therapeutic measures might prove of some benefit to the child, or at least will do no harm.

AMENTIA IN OLDER CHILDREN
Epileptic Idiocy

In discussing epilepsy (see p. 651) attention has been called to the severe mental impairment following and often accompanying recurrent epileptic attacks. In a great many cases the fits and the idiocy are based upon the same pathologic condition of the brain. G. H.

Savage remarks that the epileptic idiot is the drollest inhabitant of the idiot asylum. He is often wild, untractable, and irritable, many of the symptoms resembling those of ordinary insanity. The management of epileptic idiocy is the same as in epilepsy (*q. v.*). As the condition is practically hopeless, there need be less conservatism in advocating operative interference.

Imbecility

Imbecility is closely related to idiocy, and is based upon some inherent mental privation which no amount of education can entirely overcome. It may be the result either of congenital or acquired structural cerebral derangement consecutive to febrile affections or endocrine

Fig. 226.—Feeble-mindedness in a boy eight years old following an attack of encephalitis; he is suffering also from slight left hemiplegia. His mental age is that of a boy four years old.

disturbances. The condition is usually not detected until the child goes to school, when it is found that as compared with the normal pupil he is backward in understanding and reasoning, though he may be singularly developed in special directions, *e. g.*, memory, mechanical aptitude. Further observation reveals also that the imbecile is ex-

ceedingly emotional, easily irritated and appeased with difficulty, shows an irresistible passion to lie, steal and play truant, and that long before maturity his sexual inclinations are in the highest state of depravity. His moral decrepitude increases from year to year, and may range from theft, arson and rape to homicide and suicide.

Imbeciles should be placed under the control of experienced pedagogues, preferably in some lonely country place.

Binet-Simon Test of Mental Development*

It is now possible by means of this scale to determine fairly well the mental age of children. There will be much less indefiniteness regarding the terms used in designating the types of feeble-mindedness.

Thus, the term "idiot," which is technically restricted to those who cannot talk, corresponds to the mental age of one and two years.

The term "imbecile," which includes persons who understand spoken language and talk with varying degrees of fluency, corresponds to the mental age of three, four, five, six, and seven years.

The "moron" is one who, in addition to using spoken language, is capable of learning to read and write, and he corresponds to the mental age of eight, nine, ten, and eleven years.

The feeble-minded are persons who include all three groups and hence correspond to the mental age of from one to twelve years. A person having the mental age of twelve may be retarded, but is not feeble-minded, and technically the retarded would be persons who have the mental age of twelve to fifteen years and who do not get beyond it.

Mental Age 5 Years†

Place two boxes weighing ¼ and ½ ounce respectively on the table before the child, leaving a space of 2 inches between them, and say, "You see these two boxes? Tell me which is the heavier. Repeat using boxes weighing ½ and ¾ of an ounce, and repeat again, using first pair.

Place an oblong card on the table before the child and place also, nearer to the child, two triangular cards formed by cutting another card like the first one in two, along a diagonal. Place these two triangular cards in such position that their hypothenuses form a right angle one with the other, then say to the child, "Put these two pieces together so that they will form one card like this," (indicating the oblong card). If the child turns over one triangular piece without noticing it, it is permissible to begin again.

---

*The tests are modified to correspond to the more advanced intelligence of modern children.
†The mental tests for children under five years are given on p. 705.

Ask child—

"Is it morning now?" "Is it afternoon?" "What is a fork?" "What is a table?" "What is a chair?" "What is a horse?" "What is a mama?"

If some use of three of the objects is mentioned the response is considered correct.

Draw a diamond figure with ink and ask the child to copy it, giving him pen and ink for the purpose.

Place 13 pennies in a row on the table before the child and say, "Count these pennies for me, pointing to each one as you count it."

Mental Age 6 Years

"Say after me, 4, 7, 3, 9, 5, and repeat it yourself."

Draw a square 3 to 4 cm. in diameter with ink and ask the child to copy it, giving him pen and ink to do so.

Place one two cent and one one cent stamp on the table before the child, and then ask him to count how much they would all cost.

Have four pieces of colored paper, red, blue, yellow, and green. Point to each, asking, "What is this color?"

How many fingers have you on your right hand?

Mental Age 7 Years

(a) "Do you know what paper is?" "Do you know what cardboard is?" "Are they alike?" "In what way are they not alike?"

(b) "Have you ever seen a fly?" "Have you ever seen a butterfly?" "Are they alike?" "In what way are they not alike?"

(c) "Do you know wood when you see it?" "Do you know glass when you see it?" "Are they alike?" "In what way are they not alike?" *Two satisfactory answers required.*

"I want you to count backward from 20 to 0. Like this—20—19—18." *This must be accomplished in* 30 *seconds. One error allowed.*

"What day is to-day??" "What date is it?"

"Listen well and repeat what I say: 3-8-5-7-1; 9-2-7-3-6; and 5-1-8-3-9." *One group given at a time.*

Mental Age 8 Years

Show the child successively a penny, a dime, a dollar, a quarter, a nickel, a half dollar, a two dollar bill, a ten dollar bill, a five dollar bill. Ask, "What is this?" with each.

In a pile before the child place the following coins: Ten pennies, two nickels, two dimes, one quarter, one half dollar. Then propose a game

of storekeeping, the child to keep the store and use the pile of money to make change, the experimenter to be the customer. Add some articles for sale. Then buy something for four cents. Give the child a quarter and require the change.

"Name the months of the year in order." *One error allowed, time 15 seconds.*

"If you were going away and missed your train, what would you do."

"If one of the boys should hit you without meaning to, what would you do about it?"

"If you broke something belonging to some one else, what would you do about it?" *Two good responses required.*

Mental Age 9 Years

Place on the table before the child five boxes weighing 3, 6, 9, 12 and 15 grams respectively. Say to him, "These little boxes all weigh different amounts. Some are heavier and some lighter. I want you to place the heaviest here and by its side the one which is a little less heavy and then the one a little less heavy and the one still a little less heavy, and finally here the lightest." *Three trials made, the boxes mixed after each. Two successes in three are required.*

"I am going to show you two drawings† and after you have looked at them I shall take them away and ask you to draw them from memory. You must look at them closely because you will have them for ten seconds and this is a very short time."

Full credit is given if the whole of one drawing and half of the other is reproduced exactly.

"I am going to read you some sentences; in each one of them there is something foolish or absurd. You listen carefully and tell me each time what it is that is foolish."

(*a*) "An unlucky bicycle rider fell on his head and was instantly killed; they took him to the hospital and fear that he cannot get well." After a pause—"What is foolish in that?"

(*b*) "I have three brothers, Paul, Ernest, and myself"—"What is foolish in that?"

(*c*) "The body of a young girl cut into 18 pieces was found yesterday. People think that she killed herself."—"What is foolish in that?"

(*d*) "There was a railroad accident yesterday, but not a serious one, only 48 persons were killed."—"What is foolish in that?"

(*e*) "A man said: 'If I should ever grow desperate and kill myself

†Simple figures, *e. g.*, table, chair.

I should not use Friday for the purpose because Frdiay is an unlucky day and might bring me unhappiness.' "—"What is foolish in that?"
Correct solution of three of the five statements required.

MENTAL AGE 10 YEARS

(*a*) "If you were delayed on your way to school, what would you do about it?"

(*b*) "Before taking part in something very important what would you do?"

(*c*) "Why do we more easily pardon a bad act done in anger than a bad one done without anger?"

(*d*) "If some one should ask your opinion of one whom you did not know very well, what would you say?"

(*e*) "Why should we judge a person by his acts rather than by his words?"
Two errors allowed.

Write the words *Paris, fortune, stream.* Show them to the child, reading them to him several times. Then give him pen and ink and tell him to write a sentence containing all three of these words.

MENTAL AGE 11 YEARS

"I want you to say just as many words as you can in three minutes. Some boys say as many as two hundred. Now you must try and see how many you can think of."
Sixty words the minimum accepted.

"What does charity mean?" "What does justice mean?" "What does kindness mean?" *Two correct answers required.*

"Find the sentences which these words make. Fix the words in their proper order."

(*a*) At-country-we-for-started-hour-an-the-early.

(*b*) Teacher-I-to-my-exercise-asked-my-correct.

(*c*) Defends-a-his-dog-master-good-bravely.

The intelligence of a child is judged not only by the answer he gives, but also by the way he gives it, and the manner in which he goes about it. Some children, although very bright, may be very careless in listening to the question and give an answer which, although not correct, is nevertheless very sensible. Moreover it is well to bear in mind the fact that some children may be somewhat deficient in certain directions (*e. g., drawing*) and yet perfectly normal as regards their general intelligence.

Mental Affections in Older Children

Exempting hysteria, (*q. v.*) epileptic idiocy and imbecility, mental affections in children under twelve years of age are very rarely met in daily practice, hence no effort will here be made to dwell upon the subject very extensively. Attention, however, will be directed to the more common, though very rare, mental diseases occurring in children approaching puberty and adolescence.

Dementia

Acute dementia is rare in children under twelve years of age. It is apt to follow severe infectious diseases, such as typhoid fever, influenza, or scarlatina, or sudden shock and mental and physical overexertion. It is manifested by gradual weakening of the mind,

Fig. 227.—Dementia precox in a girl thirteen years old. Note also cystic degeneration of the thyroid gland.

characterized by loss of memory, lack of power of attention, interest, and curiosity, and tendency to stupor. After weeks or months of rest, ample nutrition and tonic treatment there is usually a progressive return of the intellect and gradual recovery. More rarely it terminates in permanent weakmindedness.

Dementia Precox Katatonia Hebephrenia

This mental affection is usually encountered in children over ten years of age, and especially in girls at the period of puberty. It usually begins

with a prodromic stage of depression and apathy during which the child loses interest in her school work, and complains of divers imaginary ailments. Loss of memory, especially for recent events, and gradual, progressive, intellectual enfeeblement form characteristic symptoms. The stage of apathy is soon followed by one of anxiety and hallucinations or outbreaks of emotional excitement, silly and hilarious in nature. As the disease advances the condition is often complicated by manifestations of mania with a marked tendency to destructiveness and violence, occasionally also by attacks of stupor, catalepsy, affection of speech, refusal of food, convulsive movements, etc.

In favorable cases the mental disturbance gradually subsides within a few weeks or months, often leaving behind symptoms of imbecility. In unfavorable cases the disease passes into a state of total idiocy. Little if anything can be done to influence the course of the affection.

Illustrative Case (Fig. 277).—When I first saw her she was thirteen years old. She was five feet six inches in height, and slender in build, weighing one hundred and twelve pounds. Her general health was good and her heart's action slow and regular. Her menstruation had not set in and her mammary glands were not developed. Her head was large and covered by a fair supply of normal hair. Her eyes were large, but not bulging, and her facial features seemed normal, when she was not laughing—which latter was rarely the case, especially when spoken to. When addressed she would invariably grin (that peculiar idiotic grin) or laugh aloud for several minutes at a time, open her mouth very wide and show a set of ugly, big, blackish brown, partially decayed, crooked teeth. Examination of the neck revealed a large, elastic, cyst-like swelling, spreading out as a broad goitrous mass, especially to the right. According to the mother the tumor developed gradually within about two years previous to my examination. The family history was apparently negative. The parents were hard-working, healthy people, and their two other children were well. The patient's mental condition was supposed to have been quite good up to ten years of age. At about that time it was noticed that she lost interest in her school work, became slovenly and forgetful and very "nervous." As weeks and months passed by her feeble-mindedness grew more and more pronounced, so that on coming under my observation I found her essentially idiotic. As already stated, when addressed she would grin and laugh; when questioned about something, she would turn to her mother and partly repeat what the mother had to say; she was unable

to add together, for example, two and two, and had no idea of where she lived. She was extremely restless and disturbed by the slightest commotion, and like a frightened baby was closely clinging to her mother's side. I put her on slowly increasing doses of thyroid extract, but it had no beneficial effect upon her feeble mentality—her condition remained stationary for several months. I lost track of her for about six months thereafter, when one day I read in the daily press that on being sent to a grocery store across the street she had lost her way back home and was picked up by a policeman the following day, exhausted from hunger, thirst, and fatigue, wading knee-deep in the swamps of Westchester and unable to give any information as to her name or place of residence. After a few similar escapades she was finally committed to an insane asylum.

Dementia Paralytica

Dementia paralytica, which is very uncommon in children and usually based upon hereditary syphilis, presents identical symptoms as in adults. Thus, tremor, slurring speech, pupillary inequality, ataxia, trophic changes, and paresis; gradual loss of intellect with development of unsystematized ideas of self-importance. The course of this form of dementia is chronic (several years) and invariably ends fatally. Slight improvement may occasionally be observed from cautious use of mercury and the iodids.

Melancholia

Mental depression is not rarely observed in children from ten to fifteen years of age and sometimes in younger ones. The child refuses to play, laments, and cries, broods over imaginary wrong acts and occasionally falls into paroxysms of rage. Melancholia not rarely leads to attempts of self-destruction.

The prognosis of this affection is fairly favorable (after weeks or months), some cases, however, may proceed to mania or even dementia.

Rest and good food are essential in the treatment.

Mania

In contrast to melancholia, mania is characterized by acceleration of every physical and mental activity. Thoughts and impulses follow one another with unusual rapidity. The patient talks, rages, screams and tries to destroy everything in sight. She also suffers from hallucinations and delusions of greatness. While mania

often ends in recovery after from six to twelve months, it also shows a great tendency to recurrence or to alternate with attacks of melancholia—circular insanity—in which event the prognosis is very bad.

The treatment, in addition to rest and proper nourishment is symptomatic—calming of the excitement by means of hyoscine hydrobromate, and other hypnotics. Luminal, gr. $1\frac{1}{2}$ twice a day will be found exceedingly useful as a general nerve sedative.

Mental affections in older children are best treated in sanitaria, away from friends and relatives. As the majority of them refuse to eat, and as ample nutrition is essential to recovery, we are often forced to feed these patients by a stomach tube introduced through the nose. Of course, for this purpose only liquid food is available, such as rich milk, fermented milk, broths and fruit juices. Medicines also may be given in this manner.

CHAPTER XIV

DISEASES OF THE SKIN

Skin affections of children like those of adults may be classified into systemic and local. To the former class belong chiefly the large group of exanthemata; the rashes arising as a result of faulty metabolism and autointoxication, including the different forms of purpura, erythema and drug eruptions; the syphilides and tuberculous lesions and the obscure dermatoneuroses. The local skin diseases embrace the local parasitic affections, the lesions following mechanic, traumatic, thermic and chemic irritations.

Since the greater number of systemic morbid skin manifestations have received due consideration in connection with the underlying diseases, we shall here limit our discussion to the skin eruptions which yield principally to local treatment.

Eczema

Eczema in children is usually observed in subacute or chronic form. It ordinarily begins with localized, more rarely diffuse, redness of the skin, slight edema, burning and itching. The condition is soon

Fig. 228.—Seborrheic eczema of head and face.

aggravated by the appearance of papules, vesicles, and pustules, and, if not promptly responding to treatment, by scabs, scales and fissures.

Eczema may remain localized, especially on the face and head, or become generalized. Eczema of the face and head is usually seen in

young infants, and is very refractory to treatment. In its typical form, the eruption of eczema faciei is generally spoken of as "crusta lactea," and consists of more or less coherent scabs of greenish or blackish-brown color, here and there interrupted by areas of red, moist ("weeping surface") and excoriated skin. From the face the eruption usually extends to the forehead, ears and head (eczema or seborrhea capitis). After prolonged duration the hair loses its luster, becomes thin and short, and the adjacent glands are painful and swollen, and often the seat of a pustular eruption as a result of scratching and secondary infection.

The course of eczema is very tedious. It may last weeks, months, or years. Improvement often alternates with aggravation of the condition. This is true especially of eczema accompanying constitutional derangement, e. g., gastrointestinal intoxication (see "Exudative Diatheses." p. 521). The duration of the disease is often prolonged by infection of the diseased as well as healthy areas with divers parasites during the act of scratching.

Treatment.—The success in the management of eczema, depends greatly upon the ease with which the underlying causes can be prevented or removed. The infantile skin being very delicate and vulnerable, it is essential to avoid its undue exposure to mechanical (scratching; woolen, rough underwear, etc.), thermal (excessive heat or cold, also direct action of the sun, etc.), and chemical (rubefacients, irritating soaps, urine, acrid discharges, etc.) irritation. The diet should be bland and regulated as to the time of feeding and its quantity. In protracted cases the Allergy test (q. v.) is often helpful in eliminating the toxic etiologic agent of the disease. Where microscopic and chemic examinations of the stools show inability to assimilate fats or carbohydrate, these must be either restricted or entirely eliminated. Sometimes good results are obtained from removal of sugar from the dietary. Plenty of water is often helpful. Constipation should be promptly remedied. Cleanliness of the skin and everything coming in contact with it should be insured.

The active treatment of eczema should be regulated in accord with the stage of the disease. While the skin is highly inflamed, all sorts of irritation should be interdicted. Tub bathing of the entire body should be discontinued for a time, first, because of the tendency of water to irritate the denuded skin, and, secondly, in view of the possibility—particularly in eczema due to external parasitic infection—of conveying the disease from one portion of the skin to the other. The healthy parts of the body, however, should be kept scrupulously clean by frequent sponging followed by careful drying.

The following soothing and protective ointment employed with great success at the New York Post-Graduate Hospital, will be found invaluable in the great majority of acute or subacute cases:

> ℞ Zinci oxidi,
> Pulveris cretæ ....āā ʒiv | 16
> Mix, and add with constant stirring:
> Olei lini (hot),
> Liq. plumbi subacet. dil. ....āā ʒij | 8

The ointment is applied once or twice a day thickly over the affected areas and covered by sterile gauze held in place by means of a bandage. Scratching of the skin should be prevented by mechanical means, such as celluloid armlets, and the like. Excoriated surfaces often heal promptly after painting with a 2 per cent solution of nitrate of silver. In subacute cases Dunn recommends the following ointment:

℞ Acidi Carbolici	gr. x	0.60
Hydrargyri Chloridi Mitis	gr. xv	1.00
Amyli		
Zinci Oxidi	āā ʒi	4.00
Vaselini	ʒi	30.00
M. Ft. Ung.		

After the inflammation subsides and scales and crusts firmly adhere to the skin, the soothing ointments are gradually replaced by those of a stimulating nature. The crusts are softened with carbolized oil (1 to 100), and gently removed. The hairy portions of the body are carefully shaved and cleansed with carbolized oil. After giving the affected skin a few hours rest we apply one of the following preparations:

℞ Acidi salicylici,		
Bismuthi subgal.	āā gr. xx	1.3
Thymolis	gr. v	0.3
Pulveris amyli	ʒiij	12
Ung. hydrargyri ammoniati	ʒij	8
Ung. zinci oxidi	q.s. ad ʒij	60

℞ Resorcini	gr. xx	1.3
Acidi carbolici	gr. x	0.65
Olei cadini	m xx	1.3
Sulphuris precipitatis	ʒij	8
Ung. petrolati	q.s. ad ʒij	60

High intestinal irrigation once a day with a quart or two of plain water or with the addition of a 2 per cent of bicarbonate of soda is

useful in all cases. In gastric hyperacidity carbonate of magnesium (gr. xxx, once a day) acts well. Obese children suffering from obstinate eczema with dryness of the skin often do well on minute doses of thyroid extract. Finally, it is worth remembering that protracted eczema is occasionally a manifestation of hereditary syphilis, and responds promptly to the exhibition of mercury and the iodides.

Urticaria

(Hives, Nettle Rash)

Urticaria is characterized by a multiform eruption of whitish, pinkish, or reddish color upon different portions of the body, which is sudden in appearance and disappearance, and shows a tendency to repeated recurrences. The eruption may consist of circular or spiral elevations ("wheals"), papules, vesicles, or hemorrhagic spots, and is generally associated with intense itching and stinging. It is frequently preceded and accompanied by gastric and nervous disturbances and rise of temperature.

Recurrent urticaria is prone to leave behind marked pigmentation of the skin or to terminate in *prurigo,* a very chronic skin affection manifested by dryness, hypertrophy and pigmentation of the skin and inflammation of the neighboring glands.

Treatment.—Since in the majority of instances, urticaria in children is the result of faulty feeding, especially of eating candies and cakes of poor quality, fish, fresh berries, and the like, it is essential to regulate the diet* (in some cases a milk diet is efficient, in others again elimination of milk may prove successful), and to clear the gastrointestinal tract of the obnoxious material. The latter is best accomplished by small doses of calomel, magnesium carbonate and sodium bicarbonate and a high enema. To relieve itching we may resort to warm baths with bicarbonate of soda ($\frac{1}{2}$ to 1 pound), sponging of the body with vinegar followed by glycerine, or to the following preparations:

R	Thymolis	gr. v to x	0.3 to 0.65
	Ung. aquæ rosæ	℥j	30
M. S.—P. r. n.			

R	Aquæ ammoniæ		ℨss	2
	Aquæ hamamelidis		℥iij	90
M. S.—*Not* to be used over abraded portions of the skin.				

*See "Food Idiosyncrasy," p. 87.

℞	Acidi carbolici	℥ss	2.00
	Zinci oxidi	℥ss	15.00
	Glycerini	℥ii	8.00
	Aq. calcis	℥viii	240.00
	M.		

S.—Apply several times a day and allow to dry on skin.

Intertrigo

(CHAFING)

This affection occurs with predilection in localities where opposed body surfaces rub against each other, and in the "napkin region." It is the result of irritation of the skin by acrid secretions or excretions (sweat, diarrheal stools, acid urine, purulent discharges, etc.), excessive heat or moisture. Intertrigo usually begins with simple erythema. At this stage it readily yields, in addition to removal of the etiologic factors, to the application of a dusting powder of:

℞	Zinci stearatis	℥iv	15
	Bismuthi subnitratis	gr. xv	1
	Amyli	℥j	30

and the separation of the opposed surfaces by thin layers of absorbent cotton or old, clean linen cloths. As the disease advances, the skin becomes glossy, moist, sticky, and denuded of the epidermis, and the seat of papules, abscesses and ulcerations. In this condition intertrigo is very refractory to treatment, often demanding a complete change in the regime of the baby—beginning with its diet and ending up with its nurse. The customary daily tub bath should be replaced by a sponge bath, taking special care to keep the affected parts of the skin perfectly dry. The denuded skin should once daily be painted with a 1 or 2 per cent solution of nitrate of silver, and the entire diseased surface covered with the following ointments:

℞	Acidi carbolici,		
	Balsami Peruviani	āā m v	0.3
	Olei lini,		
	Adipis lanæ,		
	Ung. zinci oxidi	āā ℥iv	15

M. S.—To be applied several times a day after carefully cleansing (with oil) and drying the affected parts.

℞	Thymolis	gr. ii	0.13
	Dermatolis	gr. viii	0.5
	Ung. zinci oxidi	q. s. ℥ i	30
	M.		

Sig.—P. r. n.

Psoriasis

The disease is very exceptionally met in children under five years of age, but is not uncommon in older ones. It begins with minute white spots, usually upon the extensor surfaces of the elbows and knees and upon the scalp, and gradually assumes the shape of disks with tawny-red base and silvery-white scales, not rarely giving the skin the appearance of being splashed with mortar. The cause of psoriasis being obscure (it is probably of parasitic origin, though it seems to run in families), the treatment is necessarily symptomatic;

Fig. 229.—Psoriasis in a girl seven years old.

and very unsatisfactory as to ultimate cure. Internally we may try small doses of arsenic, to be continued for several months, or thyroid extract. Externally we resort to alkaline baths, and, after the removal of the scales, to an ointment composed of chrysarobin or salicylic acid and ichthyol.

℞	Acidi salicyli,		
	Resorcini,		
	Ichthyolis	āā ℥ss	2
	Ung. sulphuris	℥ij	60
	M.		
	S.—To be applied twice a day.		

℞ Chrysarobini,
    Ichthyolis   āā 3j │ 4
    Ung. petrolati   ℥ij │ 60
    M.
    S.—To be applied once or twice a day.

Herpes Zoster

(SHINGLES)

Contrary to what is observed in adults, herpes zoster in chil-
dren is rarely accompanied by severe neuralgic pain. The eruption
usually appears suddenly in the form of groups of vesicles along the
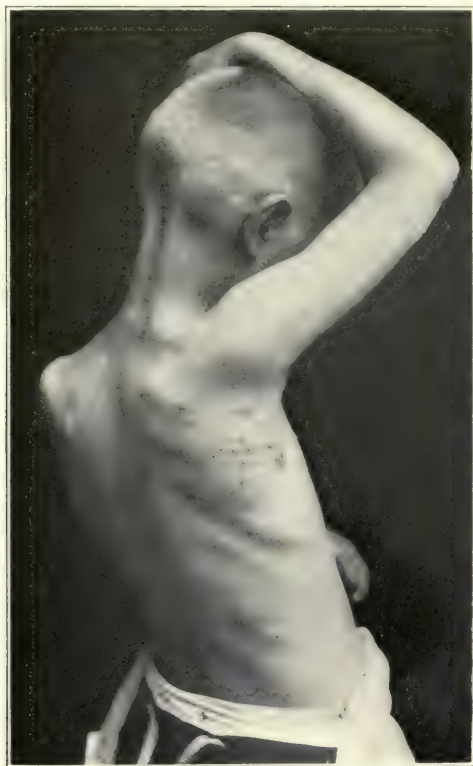
Fig. 230.—Herpes zoster.

tracts of either the intercostal or pudendal nerves, or the brachial
plexus. The vesicles remain either isolated or coalesce and form
large patches covered by yellowish-brown crusts. Different patches
often exhibit different stages of development or decline. As a rule,

the eruption is unilateral. The correlation of herpes to varicella has been spoken of on p. 395.

The course of the disease is usually completed within two weeks, except in cases leading to deep ulceration and sloughing (herpes gangrenosus), a very rare condition, usually the result of secondary infection. **Treatment** consists of local application of a dusting powder or ointment composed of stearate of zinc with or without 2 per cent of bismuth subnitrate or subgallate. Occasionally the nerve pain calls for some anodyne, *e. g.*, sodium salicylate and codeine.

Miliaria; Lichen Strophulus

(Prickly Heat)

This very common affection in infants, especially during first dentition (corresponding with the period of excessive sweating which forms a symptom of rachitis) appears suddenly upon the face, trunk, and extremities, either as discrete papules or vesicles from a pinhead to half a pea in size, or in groups upon a slightly reddened infiltrated base. It is produced by all sorts of external or internal irritations (heat, rough flannel underwear, overfeeding, etc.), and readily yields to attention to these causes, and the administration of mild laxatives. The slight itching may be relieved by cool, alkaline or bran baths, and sponging of the body with Dobell's solution. Prickly heat occurs also in older children during the summer months and is best treated by cool sponging and application of mentholated stearate of zinc.

Ecthyma

(Pseudofurunculosis)

It consists of pea- to bean-sized, flat pustules surrounded by a red zone. The lesions are situated chiefly upon the thighs, legs, shoulders and back, and are frequently associated with eczema, probably produced by infection of the eczematous lesions during the act of scratching. More rarely, they are met in the newborn as a result of lack of cleanliness (infection with dirty fingers, cloths, etc.).

Occasionally the pustules enlarge gradually and burst, leaving behind deep ulcers which heal very slowly with scar formation. These are prone to occur in ill-fed, scrofular or otherwise seriously diseased children, and may sometimes end fatally as a result of gangrene of the skin.

Simple ecthyma usually responds to hot baths (with boric acid ℥ii), antiseptic ointments, or cautious sponging of the affected parts of the body with the following:

℞ Etheris,
Tr. saponis viridis   āā ℥j | 30

Large pustules should be treated by incision and antiseptic dress-
ings. (See "Scrofulosis," p. 460.) Each pustule should be covered
with zinc adhesive plaster to prevent carrying of the pus to other parts
of the body.

Impetigo Contagiosa

The favorite seat of impetigo is the face, hands, and scalp, but the
other portions of the body are not exempt from the inoculation.

The eruption begins as small groups of minute vesicopapules which
soon burst and dry up into yellowish crusts. When the crust has
lasted for some time, its surface becomes slightly lamellated and its
edge detached, the crust then presenting the appearance as if "stuck

Fig. 231.—Impetigo contagiosa of an unusually severe type. (Courtesy of Dr. L. W.
Ketron.)

on" to the healthy skin. The surface beneath the crust is raw and
suppurating.

If further autoinoculation of the disease is prevented (by covering
with gauze and zinc adhesive plaster), impetigo usually heals spon-
taneously in about ten days. Otherwise, by the development of new
lesions, it may persist for several weeks.

Treatment.—In view of the highly contagious nature of the dis-
ease and its tendency to run in epidemic form through schools or

asylums, it is imperative to isolate all those children who are suffering from this disease and to employ active therapeutic measures to eradicate it.

This is readily accomplished by means of local antisepsis. After softening the crusts with warm carbolized sweet oil (1 per cent), and removing them, and thoroughly washing the diseased surface with green soap, the spots are touched up with a 2 to 5 per cent solution of nitrate of silver, and covered over with sterile gauze and adhesive plaster. This treatment is repeated for a few days and followed up with a 25 per cent ichthyol in a sulphur ointment.

Simple impetigo differs from the contagious variety by its lesions being pustular from the beginning and by showing no tendency to coalesce in large patches and to spread to other portions of the body. There is no history of contagion.

Pediculosis Capitis

(HEAD LICE)

The favorite seat of the head louse is the occipital portion of the scalp. In cases where the hair is thick and the parasites are few in

Fig. 232.—Pediculosis capitis, showing ova on hairs. (Courtesy of Dr. J. E. Lane.)

number and hence not easily seen, their presence can readily be surmised by the existence of ova (nits), which are firmly attached to the shafts of the hair. The lesions produced by pediculi resemble those of eczema of the head—pustules, scabs, matting of the hair, intense itching, and marked enlargement of the glands of the neck.

Treatment.—Children affected by pediculosis should be isolated for a few days until the disease is cured. The hair should be clipped, the scalp thoroughly cleansed with the tincture of green soap and then dressed with a cloth dipped in petroleum or the tincture of larkspur (delphinium). A few such dressings usually suffice to effect a cure. After removal of the pediculi the scalp should be cleared of its remaining eruption by an antiseptic ointment (*e. g.*, unguentum sulphuris).

Pediculosis Corporis

(Body Lice)

Body lice are seldom seen in young children. They give rise to red dots, itching and scratch marks. The diagnosis is settled by finding the parasite in the clothing or on the body of the child.

Treatment.—The treatment consists in destroying or baking the infested garments, scrubbing the child's body with green soap, and the application of a zinc and sulphur ointment until the eruption has entirely disappeared.

Pediculosis Pubis

(Crab Lice)

This skin affection is of diagnostic interest, principally because of the power of the crab louse to infest in addition to the hair of the pubis, abdomen, chest and axilla, also the eyebrows and eyelashes, in the latter case giving rise to a clinical picture resembling blepharitis.

The insect succumbs rapidly to the effects of mercury ointment:

℞	Ung. hydrarg. nitratis	ʒj	4
	Ung. petrolati	ʒiij	12
	M.		

S.—Externally.

Scabies

(The "Itch")

The eruption of scabies is localized chiefly in places where the skin is thinnest, *i. e.*, the hands, the folds between the fingers, the flexor

surfaces of the wrists, the anterior folds of the axilla, also the back and lower extremities. The characteristic skin lesion of scabies is the irregularly shaped, brownish-black ridge (cuniculus or burrow), the result of the burrowing process of the Acarus or Sarcoptes scabiei. The latter is the cause of scabies and can readily be demonstrated microscopically in the scrapings of the cuniculus. As the disease advances, it frequently spreads over the entire body and gives rise to
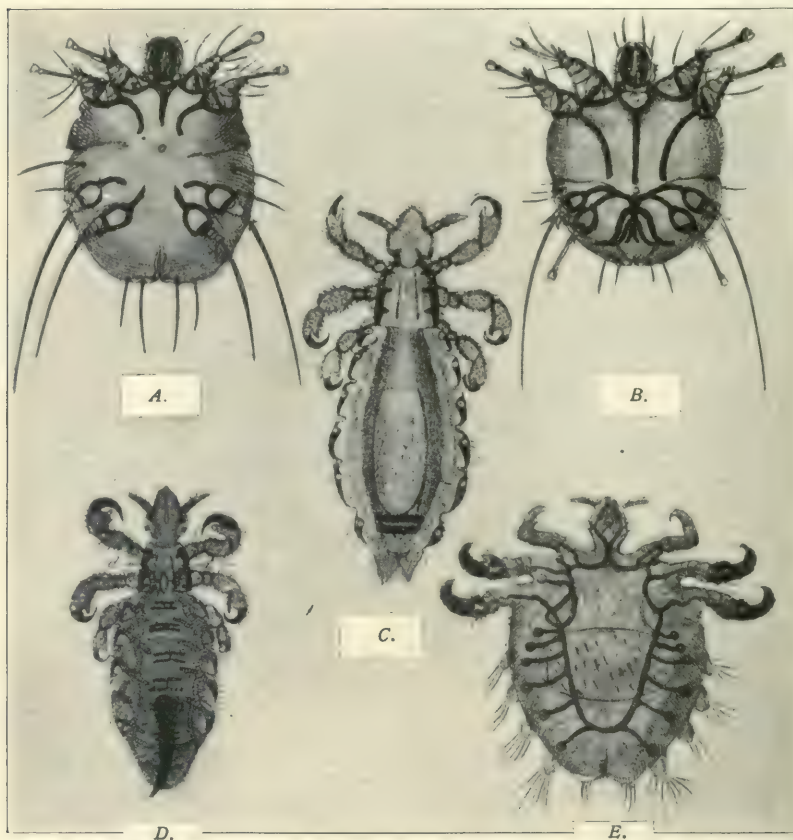
Fig. 233.—Animal parasites, *A*, acarus scabiei, female (ventral surface); *B*, acarus scabiei, male (ventral surface); *C*, pediculus corporis; *D*, pediculus capitis; *E*, pediculus pubis. (Sutton: Diseases of the Skin.)

a multiform eruption, consisting of papules, vesicles, pustules, and hemorrhagic spots (scratch marks). It is accompanied by violent itching, which is worse at night, when the patient is warm in bed.

Treatment.—As the disease is highly contagious (conveyed through close bodily contact, clothes, underwear and bedding), it is advisable

to restrict the patient from too close mingling with other members of the family or outsiders. The patient's clothes, bed sheets, towels, etc., should be boiled and the other unwashable articles thoroughly disinfected. Furthermore, all inmates of the house should be examined and, if necessary, treated for scabies, lest the disease will recur through renewed infection. The active treatment of scabies varies with the stage of the disease. Incipient scabies responds promptly to a few hot baths, thorough scrubbing of the affected skin with soft green soap and the inunction of sulphur ointment with 1 per cent carbolic acid. The management of advanced scabies with the same therapeutic measures is not quite as satisfactory. A number of remedies (strong ointments of carbolic acid, naphthol, creolin, etc.) have

Fig. 234.—Scabies, in an infant. (Richard L. Sutton.)

been suggested for such cases, but owing to their irritating qualities (upon the skin and kidneys) should be used with caution. The following combination will probably be found to do well in the majority of cases:

℞ Mentholis,
   Pulv. camphoræ   āā gr. x | 0.65
   Olei cadini,
   Balsami Peruviani   āā ʒj | 4
   Ung. sulphuris   q.s. ad ʒij | 60
   M.
S.—To be applied in the evening after a hot soap bath.

For the relief of itching we may also try the following ointment:

℞	Mentholis	gr. v	0.3
	Olei Anisi	m. xv	1.0
	Ung. Petrolati	℥ i	30.0
	M.		

Sig.—Apply once or twice daily after soap bath.

Tinea Trichophytina Capitis

(Ringworm of the Scalp, Herpes Tonsurans)

Ringworm of the scalp is due to the trichophyton fungus. It is highly contagious and often spreads with great rapidity and perti-
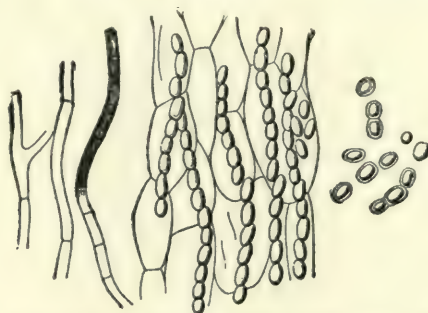
Fig 235.—Trichophyton tonsurans—threads and chains of spores × 400. (After Bizzozero.)
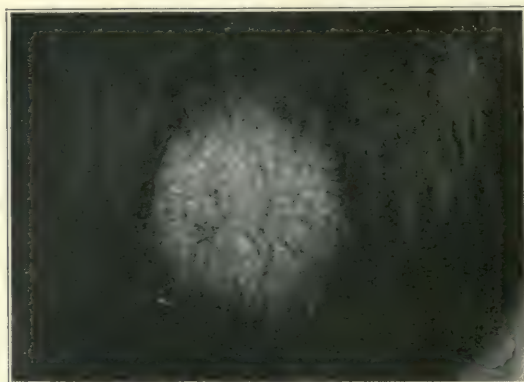
Fig. 236.—Large-spored ectothrix ringworm of scalp. (Richard L. Sutton.)

nacity in schools and children's homes where great numbers of inmates are crowded in comparatively small rooms.

The eruption consists of ring-shaped, slightly elevated, scaly, reddish, grayish, or greenish-yellow patches. The hair over the af-

fected areas becomes brittle and loose and falls out, leaving behind bald shiny spots.

At times the eruption is accompanied by severe local inflammation and exudation of a yellowish, viscid or gelatinous secretion—a condition generally described as *tinea kerion.*

Fig. 237.—Tinea tonsurans. (J. V. Shoemaker.)

Treatment.—In the treatment of ringworm of the scalp it is essential not only to prevent spreading of the disease from one child to the other, but also, to prevent autoinoculation from one part of the scalp to the other. This is best accomplished by sterilization (before and after using) of the hair clippers, scissors, combs, etc., and thorough scrubbing of the scalp with the tincture of green soap twice daily, and immediately after a hair cut.

In an epidemic at an orphan asylum comprising nearly 400 cases of ringworm of the scalp, I found the following method of treatment exceedingly serviceable:

℞ Acidi carbolici,		
Olei petrolei	āā ℥ij	65
Tinct. iodini,		
Olei ricini	āā ℥iiiss	110
Olei rusci	q.s. ad ℥xvj	500

DISEASES OF CHILDREN

After clipping the hair close to the scalp this mixture is applied over the entire scalp—more thickly over the affected spots—by means of a painter's brush, once a day for five successive days. On the sixth day it is wiped off with a rag dipped in plain olive oil; now the hair is clipped again and the scalp washed thoroughly but gently with green soap and a soft nailbrush, care being taken that all the scales and loose hair covering the scalp are removed. As a rule, no epilation is necessary. On the seventh day the mixture is reapplied as thickly as before and the whole process repeated regularly for three or four successive weeks, the length of time depending on the severity of the case. New hair will now be found to appear, and no trichophyton fungi will be discovered in the hair epilated for microscopic examination.

These procedures are followed by a few day's application of a 10 per cent sulphur ointment, and then by the use of the following preparation for about two weeks:

℞	Resorcini,		
	Acidi salicyl.	āā ℥iv	16
	Alcoholis	℥iv	120
	Olei ricini	q.s. ad. ℥xvj	500

This mixture considerably hastens the growth of the hair on the bald spots. In cases where isolation is impracticable or impossible, as often happens in private families, this resorcin mixture, daily applied, serves as an excellent substitute to prevent spreading of the affection. Tinea tonsurans is not to be confounded with *tinea favosa*, a hair affection caused by the Achorion Schönleinii, and characterized by sulphur-yellow, cup-shaped crusts or scutula, penetrated by a hair or two.

Tinea Trichophytina Corporis

(Ringworm of the Body, Herpes Circinatus)

Ringworm of the body begins as a small, scaly, circular spot which rapidly spreads peripherally and clears in the center, resembling a "ring" in shape. The rings frequently coalesce, forming serpiginous lesions.

It is a trivial eruption and promptly yields to a few local applications of the tincture of iodine, white precipitate ointment or glacial acetic acid (to be applied lightly once every other day).

Molluscum Contagiosum

Contagious molluscum is not rarely met in epidemic form in large institutions for children. The etiologic factor of the disease is as yet unknown.

The eruption appears principally upon the face, eyelids, neck and arms and consists of discrete, semiglobular, waxy-white, umbilicated, small (up to a split pea) wart-like elevations, with sebaceous contents.

It is a benign affection and readily curable by ablation of the nodule or expression of its contents, and cauterization with tincture of iodine or 5 per cent salicylic acid in collodion.

Telangiectases, Nevi and Angiomas

(Birth Marks)

Telangiectases are usually small, flat, superficial, radiating pink to bluish-red patches composed of a fine vascular net work.

Nevi are bluish-red to dark blue, flat, or elevated neoplasms of considerable size. Nevi pigmentosi may be yellow, brown, blue, black, or grayish in color; if covered by hair, they are spoken of as nevi pilosi.

Angiomas are true vascular, spongy tumors raised above the skin and containing hollow spaces filled with blood. They have a tendency

Fig. 238.—Vascular nævus. (Dr. R. L. Sutton.)

to enlarge rapidly and there is danger of ultimate sarcomatous degeneration. On the other hand, some of them undergo spontaneous evolution. Hence, in cases which do not disfigure the patient and do not make rapid progress, it is often advisable to postpone treatment as long as possible. Small birth marks frequently disappear under the application (every third day) of a solution of corrosive sublimate in collodium (6 per cent); or a single drop of a 30—volume of hydrogen dioxide is applied by means of a glass rod twice daily. If these procedures' fail, we have to resort to electrolysis or excision and ligation.

Combustio

(Burns)

In accordance with the degree of severity of the inflammation and destruction of tissue, burns are generally classified as follows:

1. **Dermatitis Ambustionis Erythematosa.**—The surface is reddened, somewhat swollen and painful and the seat of small vesicles.

2. **Dermatitis Ambustionis Bullosa.**—Blisters and bullæ of variable size, marked edema and redness of the contiguous tissues. Severe pain.

3. **Dermatitis Ambustionis Escharotica.**—Complete destruction of the integument and subcutaneous tissue, often also the muscular and fibrous tissues and even the bone. Surrounding skin blanched and markedly edematous. Sloughing of central portion.

Extensive burns in children, even if only superficial, give rise to intense constitutional symptoms, such as shock, fever, vomiting, diarrhea and sometimes convulsions. In an infant under observation a superficial burn of the neck was productive of serious edema glottidis, threatening asphyxia. Severe burns may prove fatal from shock within the first forty-eight hours or later from complicating erysipelas, duodenal ulcer, pyemia, pneumonia or tetanus.

Treatment.—Superficial burns generally improve rapidly under dressings with warm boracic acid solutions or carron oil. In order to avoid detachment of the skin on changing the dressings, it is advisable to cover the moist dressing with rubber tissue and bandage. Large blisters may be punctured with a sterile needle. After the inflammatory symptoms have subsided the following ointment will prove very efficient.

℞	Bismuthi subnitratis,		
	Resorcini	āā gr. x	0.60
	Balsami Peruviani	ʒss	2.00
	Ung. zinci oxidi	ʒi	30.00
	M. ft. ung.		

Exuberant, bleeding granulations may be reduced by daily painting with a 2 per cent nitrate of silver solution. In large and deep burns the recently recommended method of treatment by means of paraffin wax, is undoubtedly the best. The mode of application is as follows:

1. Melt the wax to fluidity, and while moderately hot,
2. Paint raw surface of burn, until thoroughly covered.
3. Apply thin layer of absorbent cotton.

4. Paint absorbent cotton with a heavy coat of the wax.

5. Cover with several layers of gauze, cotton and bandage.

6. Change dressing daily or every other day.

The wax should be melted in a sauce pan over a free flame. A large camel's hair brush is used for painting.

The constitutional symptoms should receive prompt attention. Bromides, small doses of codeine, and sterile camphorated oil hypodermically are indicated in the majority of cases. In the case referred to an ice bag seemed to do most good to relieve the edema of the glottis.

Congelatio

(Frostbite ; Chilblain)

Frostbites are quite common in children who are more or less anemic. It is usually manifested by redness, heat, itching, smarting and burning. In severe cases blisters may develop, as a rule, from scratching, and end up in indolent ulcers. Frostbites usually affect the most exposed parts, such as the hands, feet, ears, cheeks, nose and lips, and are apt to return yearly with the advent of fall and winter. Chilblains of the fingers and toes should not be mistaken for acute inflammatory rheumatism, which is a febrile affection.

In the management of frostbites due consideration should be given to the underlying constitutional debility (administration of iron and codliver oil) and proper clothing suitable for the season of the year (flannel or silk underwear, etc.). Locally the parts should be bathed in hot water, and painted with balsam of Peru or ichthyol. A very useful ointment is the following:

℞	Camphoræ		
	Creosoti	āā gr. xv	1.0
	Balsami Peruviani		
	Ichthyolis	āā ʒss	2.0
	Ung. petrolati	℥i	30.0

M. S.—To be applied once or twice a day.

INDEX

Date Due